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- Low-Dose Theophylline Treatment for Asthma
- Yes! No! Points of View on Tracheal Pressure-Triggering
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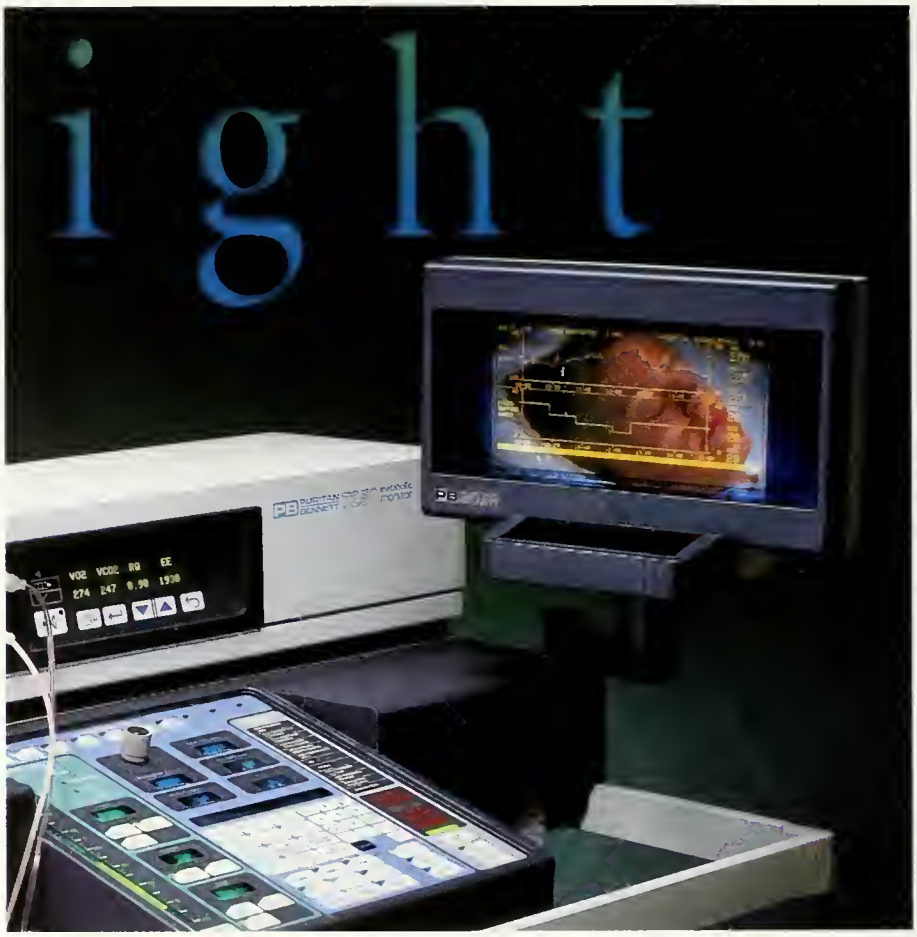

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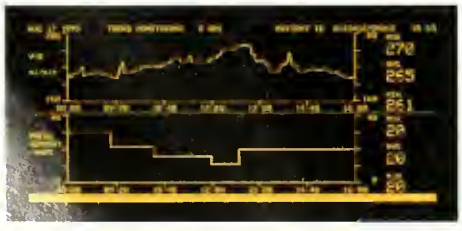

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with Rob Chatburn, RRT, and Richard D. Branson, RRT
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with James K. Stoller, MD, and Sam Giordano, MBA, RRT
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#6• *Capturing Respiratory Services Outside the Hospital Part II: Subacute Care*
with Kevin Cornish, RRT, and Sam Giordano, MBA, RRT
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519 Corrected figure
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The contents of the Journal are indexed in Hospital and Health Administration Index, Cumulative Index to Nursing and Allied Health Literature, and Excerpta Medica. Abridged versions of RESPIRATORY CARE are also published in Italian and Japanese, with permission from Daedalus Enterprises Inc.
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Editorials, Commentaries, and Reviews To Note

Tuberculosis in Children: An Update (State of the Art)—LS Inselman. Pediatr Pulmonol 1996;21(2): 101-120.

Goals of Asthma Management: A Step-Care Approach (Review)—RC Bone. Chest 1996;109: 1056-1065.

Pulmonary Administration of Vasoactive Substances by Perfluorochemical Ventilation—MR Wolfson, JS Greenspan, TH Shaffer. Pediatrics 1996;97(4):449.

OBJECTIVES: Therapeutic management of respiratory distress syndrome, pneumonia, and pulmonary hypertension includes delivery of biologically active agents to the neonatal lung. However, mechanical abnormalities of the lung, intrapulmonary shunting, ventilation-perfusion mismatching, and elevated surface tension impede effective systemic or intratracheal delivery of agents to the lung during conventional gas ventilation. The objective of this study was to test the hypothesis that perfluorochemical (PFC) liquid ventilation can be used for pulmonary administration of vasoactive drugs (PAD) and to compare these responses to those elicited with intravascular (IV) administration during tidal liquid ventilation. METHODS: Cardiovascular responses of 16 preterm and neonatal lambs to randomized doses of acetylcholine, epinephrine, and Priscoline were studied. Physiologic gas exchange and acid-base balance were maintained using previously described tidal liquid ventilation techniques. In subgroups of animals, the distribution pattern of carbon 1- and choline 14-labeled dipalmitoylphosphatidylcholine (14C-DPPC) in saline and the responses to priscoline after hypoxia-induced pulmonary hypertension and hypoxemia administered during liquid ventilation were studied. RESULTS: Dose-response curves for PAD and IV administration demonstrated progressive, dose-dependent, cholinergic responses to acetylcholine (decreased mean systemic arterial pressure [MAP] and heart rate), sympathomimetic responses to epinephrine (increased MAP and heart rate), and a-adrenergic blockade responses to priscoline (decreased MAP and mean pulmonary arterial pressure). Compared with IV administration, PAD of Priscoline resulted in a significantly greater decrease in pulmonary relative to systemic arterial pressure; this response was potentiated by hypoxia, reduced pulmonary pressures to near normal values, and improved oxygenation. The 14C-DPPC in saline was distributed relatively homogeneously throughout the lung by PAD, with 80% of the lung pieces receiving amounts of 14C-DPPC with ± 20% of the mean value. CONCLUSIONS: This study demonstrates that vasoactive agents can be delivered to the lung directly by PAD during PFC liquid ventilation. The inherent advantages of this method relate to the physical properties of PFC liquid ventilation as a vehicle (respiratory gas solubility, low surface tension ± enhancing distribution, and inertness precluding interaction) and physiological properties of the lung as an exchanger.

Low Measured Auto-Positive End-Expiratory Pressure during Mechanical Ventilation of Patients with Severe Asthma: Hidden Auto-Positive End-Expiratory Pressure—JW Leatherman, SA Ravenscraft. Crit Care Med 1996;24 (3):541.

OBJECTIVE: To describe the occurrence of low measured auto-end-expiratory pressure (auto-PEEP) during mechanical ventilation of patients with severe asthma. DESIGN: Observational clinical study. SETTING: Medical intensive care unit of a university-affiliated county hospital. PATIENTS: Four mechanically ventilated patients with severe asthma who had low measured auto-PEEP despite marked increase in both peak and plateau airway pressures. INTERVENTIONS: None. MEASUREMENTS & MAIN RESULTS: Peak pressure, plateau pressure, and auto-PEEP were measured at an early time point, when airflow obstruction was most severe, and again at a later time after clinical improvement. Auto-PEEP was measured by the method of end-expiratory airway occlusion. From the early to the late time point, there was a marked decrease in peak pressure (76 ± 7 to 53 ± 6 cm H_2O, $p < 0.001$) and in plateau pressure (28 ± 2 to 18 ± 3 cm H_2O, $p < 0.001$), but only minimal change in auto-PEEP (5 ± 3 to 4 ± 3 cm H_2O). The difference between plateau pressure and auto-PEEP decreased between the early and late time points (23 ± 1 to 14 ± 1 cm H_2O; $p < 0.01$), even though tidal volume was larger at the late time point. In 3 patients, low auto-PEEP and a large difference between plateau pressure and auto-PEEP was only seen after expiratory time was prolonged. In these 3 patients, prolongation of expiratory time resulted in a large decrease in measured auto-PEEP (14 ± 4 to 5 ± 4 cm H_2O), but a much smaller change in plateau pressure (31 ± 3 to 29 ± 3 cm H_2O). CONCLUSIONS: We conclude that measured auto-PEEP may underestimate end-expiratory alveolar pressure in severe asthma, and that marked pulmonary hyperinflation may be present despite low measured auto-PEEP, especially at low respiratory rates. This phenomenon may be due to widespread airway closure that prevents accurate assessment of alveolar pressure at end-expiration. *See the related editorial:* ***Occult, occult auto-PEEP in status asthmaticus.*** *TE Stewart, AS Slutsky. Crit Care Med 1996;24(3):379-380.*

Tracheal Gas Insufflation Combined with High-Frequency Oscillatory Ventilation—S Dolan, S Derdak, D Solomon, C Farmer, J Johanningman, J Gelineau, RB Smith. Crit Care Med 1996;24(3):458.

OBJECTIVES: To determine the efficacy of tracheal gas insufflation delivered by 2 different catheter designs on CO_2 elimination when used in conjunction with high-frequency oscillatory ventilation. DESIGN: A nonrandomized before and after trial. Each animal served as his own control. SUBJECTS: Ten mongrel dogs weighing 20.9 ± 1.9 kg. Four animals were assigned to a normal lung group and 6 animals underwent lung injury by large volume saline lavage. INTERVENTION: Permissive hypercapnia was allowed to occur by selecting oscillator settings that would lead to alveolar hypoventilation. Proximal mean airway pressure was kept constant. Tracheal gas was insufflated at 1 cm above the carina for 30-minute periods at gas flows of 5 to 15 L/min. MEASUREMENTS & MAIN RESULTS: Carinal pres-

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sure, hemodynamic parameters (cardiac output, mean arterial pressure, pulmonary arterial pressure, pulmonary artery occlusion pressure), and gas exchange parameters (P_{aCO_2}, P_{aO_2}, P_{aO_2}/F_{IO_2}, shunt fraction, $\dot{D}_{O_2}$) were measured. For the normal dogs, at catheter flow of 15 L/min, the forward thrust catheter increased carinal pressure and P_{aO_2}/F_{IO_2} by 30% ($p < 0.003$) and 105% ($p < 0.005$), respectively. The forward thrust catheter reduced P_{aCO_2} by 40% ($p < 0.04$). The reverse thrust catheter increased P_{aO_2}/F_{IO_2} by 102% ($p < 0.001$) and decreased carinal pressure and P_{aCO_2} by 44% ($p < 0.001$) and 34% ($p < 0.003$), respectively. For the injured dogs, at catheter flow rate of 15 L/min, the forward thrust catheter increased carinal pressure, P_{aO_2}, and P_{aO_2}/F_{IO_2} by 6% ($p < 0.001$), 23% ($p < 0.001$) and 24% ($p < 0.02$), respectively. The forward thrust catheter reduced P_{aCO_2} by 29% ($p < 0.002$). The reverse thrust catheter increased P_{aO_2} and P_{aO_2}/F_{IO_2} both by 11% ($p < 0.02$) and reduced carinal pressure and P_{aCO_2} by 23% ($p < 0.001$) and 18% ($p < 0.002$), respectively. CONCLUSIONS: Tracheal gas insufflation is capable of improving oxygenation and ventilation in acute lung injury when combined with high-frequency oscillatory ventilation. The addition of this second gas flow at the level of the carina raises or lowers distal airway pressure, the magnitude of which is dependent on the direction and rate of gas flow. The beneficial effects of tracheal gas insufflation may be tempered by the long-term effects of altering distal airway pressure; lowering distal airway pressure may lead to atelectasis, whereas raising distal airway pressure may lead to an auto-positive end-expiratory pressure (auto-PEEP) effect. *See the related Editorial:* ***Adjunctive Ventilation with Tracheal Gas Insufflation—Good Vibrations?—JJ Marini. Crit Care Med 1996;24(3)375-378.***

Effects of Availability of Patient-Related Charges on Practice Patterns and Cost Containment in the Pediatric Intensive Care Unit—RC Sachdeva, LS Jefferson, J Coss-Bu, G Done, D Campbell, SI Nelson, RD Feigin. Crit Care Med 1996;24(3):501.

OBJECTIVE: To investigate the effects of the availability of daily patient-related charges to health-care providers on practice patterns and cost containment in the pediatric intensive care unit (ICU) setting. DESIGN: Prospective, nonrandomized, controlled trial. SETTING: Pediatric ICU. PATIENTS: All patients admitted to the pediatric ICU during the study period. This number included a prospective control group (n = 325) and an intervention group (n = 273). These 598 patients spent 2,274 patient days in the pediatric ICU. INTERVENTIONS: The daily itemized patient charges related to diagnostic studies ordered in the pediatric ICU were made available to health-care providers during the intervention period of the study. MEASUREMENTS & MAIN RESULTS: Information was collected prospectively on patients in the control group before the intervention period. This information included data on demographics, daily severity of illness measures, daily resource consumption, intensity of nursing and medical interventions, and daily patient-related charges. Outcome information on survival and length of pediatric ICU stay was also collected. The same data were collected prospectively during the intervention period of the study. Measurements on quality assurance and morbidity were made to ensure that there was no compromise in patient care. There were no significant differences in patient demographics and diagnoses between the control and intervention groups. There was a reduction in the average daily laboratory (16.7%), radiology (9.1%), computerized axial tomography (8.5%), and pharmacy (25.1%) charges in the intervention group as compared with controls. The decreases in laboratory and pharmacy charges were statistically significant ($p < 0.0001$). The decreases in laboratory and pharmacy charges remained significant even after adjustment for severity of illness. CONCLUSIONS: The availability of patient-related charges to health-care providers can result in changes in practice patterns, producing a decrease of patient charges and an improvement in cost containment in the pediatric ICU.

Distribution and Transmission of *Pseudomonas aeruginosa* and *Burkholderia cepacia* in a Hospital Ward—G Döring, S Jansen, H Noll, H Grupp, F Frank, K Botzenhart, K Magdorf, U Wahn. Pediatr Pulmonol 1996;21(2):90-100.

Genotyping and antibiotic susceptibility testing were used to analyze *Pseudomonas aeruginosa* and *Burkholderia cepacia* strains from sink drain from 14 pediatric patients with cystic fibrosis (CF) and from hospital personnel as part of a 4-week prospective study of strain transmission in a pediatric ward. A total of 87.5% of all washbasin drains were contaminated with *P. aeruginosa* [10^2 to 10^5 colony forming units (CFU)/mL sink fluid], whereas *B. cepacia* was found only once in a sink drain. From the 8 CF patients already infected with *P. aeruginosa* upon entering the ward, we isolated 6 genotypes that were identical with strains found in sink drains of the ward. Four of the 16 members of the personnel had 1 positive *P. aeruginosa* hand culture. *B. cepacia* was never found in patients or on personnel hands. Hand washing in contaminated sinks ($\geq 10^3$ CFU/mL) led to positive *P. aeruginosa* or *B. cepacia* hand cultures. *P. aeruginosa* or *B. cepacia* embedded in sputum were transmissible by hand shaking for up to 180 minutes, whereas both pathogens suspended in physiological saline were transmissible to other hands only up to 30 minutes. Genotyping of *P. aeruginosa* revealed strain transmission from CF patients or the environment to other patients or the personnel, as well as 1 transmission from the environment to a CF patient. The ability of CF sputum to prolong survival of *P. aeruginosa* and *B. cepacia* may be important for strain transmission. The results suggest that improved hygienic measures are required to prevent routes of bacterial transmission via the hands and sink drains.

Pulse Oximetry: Accuracy of Methods of Interpreting Graphic Summaries—VM Lafontaine, FM Ducharme, RT Brouillette. Pediatr Pulmonol 1996;21(2):121.

Although pulse oximetry has been used to determine the frequency and extent of hemoglobin desaturation during sleep, movement artifact can result in overestimation of desaturation unless valid desaturations can be identified accurately. Therefore, we determined the accuracy of pulmonologists' and technicians' interpretations of graphic displays of desaturation events, derived an objective method for interpreting such events, and validated the method on an independent data set. Eighty-seven randomly selected desaturation events were classified as valid (58) or artifactual (29) based on cardiorespiratory recordings (gold standard) that included pulse waveform and respiratory inductive plethysmography signals. Using oximetry recordings (test method), 9 pediatric pulmonologists and 3 respiratory technicians ("readers") averaged 50 ± 11% (SD) accuracy for event classification. A single variable, the pulse amplitude modulation range (PAMR) prior to desaturation, performed better in discriminating valid from artifactual events with 76% accuracy ($p < 0.05$). Following a seminar on oximetry and the use of the PAMR method, the readers' accuracy increased to 73 ± 2%. In an independent set of 73 apparent desaturation events (74% valid, 26% artifactual), the PAMR method of assessing oximetry graphs yielded 82% accuracy; transcutaneous oxygen tension records confirmed a drop in oxygenation during 49 of 54 (89%) valid desaturation events. In conclusion, the most accurate method (91%) of assessing desaturation events requires recording of the pulse and respiratory waveforms. However, a practical, easy-to-use method of interpreting pulse oximetry recordings achieved 76-82% accuracy, which constitutes a significant improvement from previous subjective interpretations.

Exposure of the U.S. Population to Environmental Tobacco Smoke: The Third National Health and Nutrition Examination Survey, 1988 to 1991—JL Pirkle, KM Flegal, JT Bernert, DJ Brody, RA Etzel, KR Maurer. JAMA 1996;275(16):1233.

OBJECTIVE: To estimate the extent of exposure of the U.S. population to environmental tobacco smoke and the contribution of the home and workplace environment to environmental tobacco smoke exposure. DESIGN: Nationally representative cross-sectional survey including questionnaire information from persons aged 2 months and older (n=16,818)

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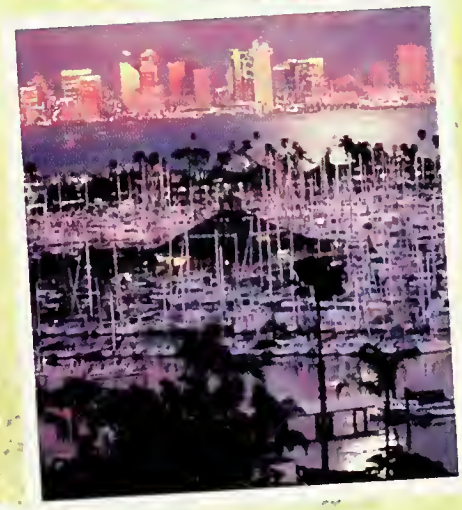

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and measurements of serum cotinine (a metabolite of nicotine) from persons aged 4 years and older (n=10,642). SETTING/PARTICIPANTS: Participants in the Third National Health and Nutrition Examination Survey, October 25, 1988, to October 21, 1991. RESULTS: Of U.S. children aged 2 months to 11 years, 43% lived in a home with at least 1 smoker, and 37% of adult non-tobacco users lived in a home with at least 1 smoker or reported environmental tobacco smoke exposure at work. Serum cotinine levels indicated more widespread exposure to nicotine. Of non-tobacco users, 87.9% had detectable levels of serum cotinine. Both the number of smokers in the household and the hours exposed at work were significantly and independently associated ($p < 0.001$, multiple regression t test) with increased serum cotinine levels. Serum cotinine levels of children, non-Hispanic blacks, and males indicated that these groups had higher exposure to environmental tobacco smoke. Dietary variables showed no consistent association with serum cotinine levels, and dietary contribution to serum cotinine level, if any, appeared to be extremely small. CONCLUSIONS: The high proportion of the population with detectable serum cotinine levels indicates widespread exposure to environmental tobacco smoke in the U.S. population. Both the home and workplace environments significantly contribute to environmental tobacco smoke exposure in the United States.

Employment Implications of Declining Tobacco Product Sales for the Regional Economies of the United States—KE Warner, GA Fulton, P Nicolas, DR Grimes. JAMA 1996;275(16):1241.

OBJECTIVE: To determine whether declines in tobacco product sales significantly reduce employment in the United States, as the tobacco industry claims. DESIGN: Computer simulation of the economies of the Southeast Tobacco region and 8 nontobacco regions of the United States, with domestic tobacco expenditures eliminated or reduced and the equivalent spending redistributed, according to consumers' normal spending patterns. We compared these results with baseline forecasts of the regional economies that include normal tobacco expenditures. MAIN OUTCOME MEASURE: Number of jobs. RESULTS: Had there been no spending on tobacco products in the United States in 1993, the Southeast Tobacco region would have had 303,000 fewer jobs. Collectively, however, the 8 nontobacco regions would have gained enough employment to completely offset losses in the Southeast Tobacco region, with every nontobacco region gaining jobs. By the year 2000, the absence of tobacco spending would mean a loss of 222,000 jobs in the Southeast Tobacco region, but a gain of 355,000 throughout the rest of the country. In the more realistic scenario of doubling the downward trend in tobacco consumption, the Southeast Tobacco region would lose 6,300 jobs in 1993 (0.03% of regional employment) and 36,600 jobs by 2000 (0.2%). The 8 nontobacco regions would gain 6,400 jobs in 1993 and 56,300 jobs in 2000, with each of the nontobacco regions gaining employment in every year. CONCLUSIONS: Contrary to the tobacco industry's claims, reductions in spending on tobacco products will boost employment in every one of the 8 nontobacco regions and will not diminish employment in the Southeast Tobacco region by as much as the industry estimates. The primary concern about tobacco should be the enormity of its toll on health and not its impact on employment.

Cost-Effectiveness of the Transdermal Nicotine Patch as an Adjunct to Physicians' Smoking Cessation Counseling—K Fiscella, P Franks. JAMA 1996;275(16):1247.

OBJECTIVE: To determine the incremental cost-effectiveness of the transdermal nicotine patch. DESIGN: Decision analytic model that evaluated the incremental cost-effectiveness of the addition of the nicotine patch to smoking cessation counseling. Costs were based on physician time and the retail cost of the nicotine patch, and benefits were based on quality-adjusted life years (QALYs) saved. PATIENTS: Male and female smokers aged 25 to 69 years receiving primary care. INTERVENTION: Addition of the nicotine patch to physician-based smoking cessation counseling. MAIN OUTCOME MEASURE: Costs (1995 dollars) per QALYs saved discounted by 3% annually. RESULTS: The use of the patch produced 1 additional lifetime quitter at a cost of $7,332. The incremental cost-effectiveness of the nicotine patch by age group ranged from $4,390 to $10,943 per QALY for men and $4,955 to $6,983 per QALY for women. A clinical strategy involving limiting prescription renewals to patients successfully abstaining for the first 2 weeks improved the cost-effectiveness of the patch by 25%. CONCLUSIONS: The findings provide support both for the routine use of the nicotine patch as an adjunct to physicians' smoking cessation counseling and for health insurance coverage of nicotine patch therapy.

Hospital Smoking Bans and Employee Smoking Behavior: Results of a National Survey—DR Longo, RC Brownson, JC Johnson, JE Hewett, RL Kruse, TE Novotny, RA Logan. JAMA 1996;275(16):1252.

OBJECTIVE: To examine the impact of workplace smoking bans on smoking behavior of employees. PARTICIPANTS: A total of 1,469 current or former smokers (intervention group) employed in smoke-free hospitals and 920 current or former smokers (comparison group) employed in non-smoke-free workplaces were surveyed to determine smoking behavior. DESIGN: This cross-sectional study is part of a larger, ongoing prospective study. The study design was quasi-experimental. We randomly selected sites consisting of a hospital and a corresponding community. Furthermore, we randomly selected subjects from hospitals and their corresponding communities. MAIN OUTCOME MEASURES: Postban quit ratio and progression along the stages-of-change continuum. METHODS: The Cox proportional hazards model was used to compare the postban quit ratio between the intervention and comparison groups. The Cochran-Mantel-Haenszel analysis of variance statistic was used to compare groups on the stages-of-change variables. RESULTS: Beginning with the smoking ban and continuing for 5 years after implementation, statistically significant differences in the postban quit ratio were observed between employees of smoke-free hospitals who were smokers and counterparts in the community ($p < 0.001$). Despite preban differences in smoking intensity, the overall difference in postban quit ratios remained significant even after multivariate adjustment for socioeconomic, demographic, and smoking intensity variables. For those sites that were 5 years postban, the quit ratio was 0.506 in smoke-free workplaces compared with 0.377 in workplaces where smoking was permitted. In all but 1 category, the intervention group was further along the stages-of-change continuum toward quitting smoking than the comparison group ($p < 0.001$). CONCLUSION: American hospitals' experiences with smoking bans, which directly affect more than 5 million workers, should be examined by other industries as a method of improving employee health. Workplace smoking bans could also be effective in saving lives, reducing health-care costs, addressing safety concerns, and decreasing operating and maintenance expenses of employers.

The Agency for Health Care Policy and Research Smoking Cessation Clinical Practice Guideline—The Smoking Cessation Clinical Practice Guideline Panel & Staff. JAMA 1996;275(16):1270.

OBJECTIVE: To summarize the Smoking Cessation Clinical Practice Guideline that provides recommendations for 3 groups of professionals: primary care clinicians, smoking cessation specialists, and health-care administrators, insurers, and purchasers. PARTICIPANTS: An independent panel of scientists, clinicians, consumers, and methodologists selected by the U.S. Agency for Health Care Policy and Research. EVIDENCE: English-language, peer-reviewed literature published between 1975 and 1994 that addresses the assessment and treatment of tobacco dependence, nicotine addiction, and clinical practice. CONSENSUS PROCESS: Four panel meetings were held over 2 years to evaluate meta-analytic and other results, to synthesize the results, and to develop recommendations. The Guideline was repeatedly reviewed and revised. CONCLUSIONS: The panel recommendations address 3 audiences. Major recommendations for primary care clinicians are to use officewide systems to

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identify smokers, treat every smoker with a cessation or motivational intervention, offer nicotine replacement except in special circumstances, and schedule follow-up contact to occur after cessation. Major recommendations to smoking cessation specialists are to use multiple individual or group counseling sessions lasting at least 20 minutes each with sessions spanning multiple weeks, offer nicotine replacement, and provide problem-solving and social support counseling. Major recommendations for health-care administrators, insurers, and purchasers are that tobacco-user identification systems be used in all clinics and that smoking cessation treatment be supported through staff education and training, dedicated staff, changes in hospital policies, and the provision of reimbursement for tobacco-dependence treatment.

Short-Term Efficacy of Ultrasonically Nebulized Hypertonic Saline in Cystic Fibrosis—PA Eng, J Morton, JA Douglass, J Riedler, J Wilson, CF Robertson. Pediatr Pulmonol 1996;21(2):77.

Progressive lung disease in patients with cystic fibrosis (CF) is caused by thick secretions, which cause airway obstruction and subsequent colonization and infection by inhaled pathogenic microorganisms. Recently, recombinant human DNase has been shown to reduce the viscoelasticity of sputum in patients with cystic fibrosis and to improve lung function. Ultrasonically nebulized hypertonic saline (HS) has been demonstrated to enhance mucociliary clearance and sputum expectoration by rehydrating airway secretions, and may therefore provide a low-cost alternative. We studied the changes in pulmonary function and symptoms in a group of patients with CF who have moderate to severe lung disease. The patients were evaluated following 2 weeks of treatment with HS in an open-label study. Subjects were randomly allocated to receive 10 mL of either 0.9% NaCl (IS) or 6% NaCl (HS). Twice daily, prior to physiotherapy, treatments were delivered by a portable ultrasonic nebulizer. To prevent bronchoconstriction, 600 mg of salbutamol were administered prior to the nebulized solutions. A symptom score was recorded, and spirometry was performed on day 0 before therapy was started, on day 14 (the last day of therapy), and on day 28 (14 days after the last treatment with either IS or HS). Fifty-two patients (32 males), with a mean age of 16.2 (range 7-36) years completed the study. There was no difference in baseline characteristics between the 2 groups. Following 2 weeks of treatment, there was a significant improvement from baseline in FEV_1 of 15.0 ± 16.0% (mean ± SD) in patients treated with HS, compared with a change of 2.8 ± 13% in those on IS therapy ($p = 0.004$). Furthermore, there was a subjective improvement in the effectiveness of chest physiotherapy as reported by those using HS ($p = 0.02$). The treatment was well tolerated. We conclude that in patients with CF, ultrasonically nebulized hypertonic saline improves lung function in a way similar to that reported for human recombinant DNase when inhaled over a 2-week period. Nebulized saline also enhances the perception of effectiveness of chest physiotherapy. *See the related editorial:* ***Hypertonic Saline in Cystic Fibrosis****—GJ Canny. Pediatr Pulmonol 1996;21:73-74.*

The Role of Sleep-Disordered Breathing in Essential Hypertension—TV Coy, JE Dimsdale, S Ancoli-Israel, JL Clausen. Chest 1996;108 (4):890.

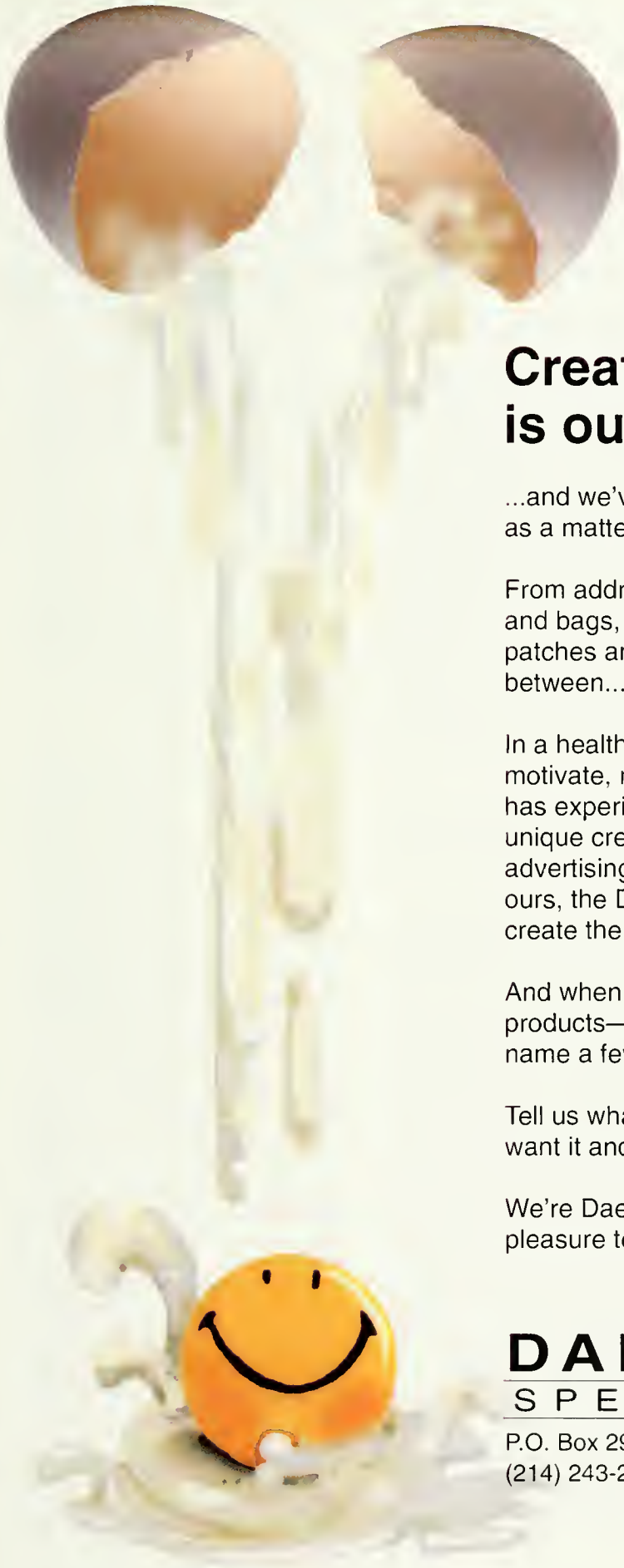
In recent years, there have been numerous reports addressing the relationship between sleep-disordered breathing (SDB) and hypertension (HTN). This study investigated the relationship between SDB and BP after controlling for age, gross obesity, and notably, antihypertensive medications. Sixty-seven men and women between 30 and 60 years of age and between 0.90 to 1.5 times ideal body weight were studied. SDB was assessed over 2 nights of polysomnographic monitoring, and BP was measured over repeated visits to the hospital. The results indicate that respiratory disturbance index (RDI) independently predicts diastolic BP (DBP), accounting for 15% of the variance in DBP ($p = 0.02$). In subjects with severe levels of SDB (RDI > 30), RDI uniquely accounted for 36% of the variance in DBP ($p = 0.003$). Interestingly, SDB was not independently related to systolic BP. The physiologic mechanisms responsible for these findings are currently being explored. *See the related editorial:* ***Hypertension and OSA: Silent Bedpartners?****—NA Collop. Chest 1996;109(4): 861-862.*

A Comparative Study of the Clinical Efficacy of Nedocromil Sodium and Placebo: How Does Cromolyn Sodium Compare as an Active Control Treatment?—HJ Schwartz, M Blumenthal, R Brady, S Braun, R Lockey, D Myers, L Mansfield, M Mullarkey, G Owens, P Ratner, L Repsher, A van As. Chest 1996;109(4):945.

Nedocromil sodium and cromolyn sodium are the only 2 currently available nonsteroid anti-inflammatory agents for treatment of asthma. Clinical differences between the 2 agents remain under continuous investigation with reports differentiating the two on the basis of atopy of the patient and reversibility of bronchoconstriction. This study investigated the efficacy of nedocromil sodium (4 mg, qid) for treatment of mild-to-moderate asthma in comparison to placebo using cromolyn sodium (2 mg, qid) as an active control treatment. Patients were primarily allergic asthmatics (with at least 15% reversibility) previously maintained on a regimen of regular bronchodilator therapy. During a 2-week run-in period, the patient's slow-release theophylline therapy was removed, and the patients were randomized to treatment after deterioration of asthma control (asthma symptom summary score of 3 for 7 of the 14 days). After 8 weeks of treatment, patients were returned to as occasion requires bronchodilator therapy, as per the 2-week baseline period. The results demonstrate that patients treated with nedocromil sodium showed statistically significant improvements during the primary time period (mean weeks 3 through 8) over placebo-treated patients as evidenced by all indexes of asthma symptoms, pulmonary function measures, and decreased bronchodilator reliance ($p < 0.05$). Patients treated with cromolyn sodium demonstrated similar improvements over placebo-treated patients. Comparisons between nedocromil sodium and cromolyn sodium showed the 2 agents to be comparable in this group of primarily allergic patients with reversible disease. Between-group differences were noted for 3 of the 13 variables (nighttime asthma, FEV_1, and forced expiratory flow rate between 25% and 75% of the FVC) in favor of cromolyn sodium when the data were pooled during the primary time period. The number of patients missing 1 or more days from work/school/regular activity due to asthma was significantly fewer compared with placebo, and favoring nedocromil sodium over cromolyn sodium. No differences were observed among the 3 treatments for adverse events. This study demonstrated that in primarily allergic patients with reversible airways disease, nedocromil sodium and cromolyn sodium are both significantly more effective than placebo for treatment of mild-to-moderate asthma.

Inhaled Corticosteroids Do Not Prevent the Development of Tolerance to the Bronchoprotective Effect of Salmeterol—S Kalra, VA Swystun, R Bhagat, DW Cockcroft. Chest 1996;109(4):953.

Twice daily inhaled salmeterol produces rapid reduction in its acute bronchoprotective effect against methacholine in patients with mild asthma. This investigation examined this effect in patients with moderate asthma who were using inhaled corticosteroids. SUBJECTS & METHODS: Eight asthmatic volunteers who required inhaled corticosteroids for control of their symptoms and who were able to withhold treatment with β-2 agonists for 4 weeks before and during the study participated in a double-blind, crossover, placebo-controlled study with 2 random-order treatment periods: inhaled salmeterol, 50 μg twice a day for 7 doses, and placebo in similar fashion, with a 7-day or greater washout between these periods. Methacholine inhalation tests were done 1 hour after doses 1, 3, 5, and 7, and then 24 hours after the last dose of the study inhaler, 10 minutes post-200 μg salbutamol. RESULTS: Baseline FEV_1 measurements before doses 3, 5, and 7 of salmeterol, ie, 12 hours after salmeterol, were significantly higher than all other baseline values. Twenty-four hours after the last dose of salmeterol, the FEV_1 was no different from that during the placebo period. The geometric mean methacholine concentration causing a 20% fall in FEV_1 (PC_{20}) following the third dose of sal-



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meterol (6.8 mg/mL) was significantly lower than after the first dose of salmeterol (12.0 mg/mL; $p = 0.031$), and this reduction of bronchoprotection persisted following doses 5 and 7. The methacholine PC_{20} 10 minutes postsalbutamol measured after the salmeterol period was significantly lower than after placebo (5.6 vs 13.3 mg/mL; $p < 0.001$). CONCLUSIONS: Tolerance to the acute bronchoprotective effect of salmeterol was significant after the first 2 doses and persisted after the seventh dose. Tolerance to the acute bronchoprotective effect of salbutamol was also significant after regular use of salmeterol for 7 doses. These effects, in subjects using inhaled corticosteroids regularly, were similar to those previously seen in patients with mild asthma using as-required β-2 agonists only, indicating that tolerance is not prevented by use of inhaled corticosteroids. *See the related editorial:* ***Long-acting β-Agonists, Tachyphylaxis, and Corticosteroids*** *—MR Sears. Chest 1996;109 (4):862-864.*

Inhalation of Single vs Multiple Metered Dose Bronchodilator Actuations from Reservoir Devices: An In Vitro Study—JL Rau, RD Restrepo, V Deshpande. Chest 1996;109(4):969.

Differences in inhalation technique with reservoir or spacer devices may affect metered dose inhaler (MDI) dose availability to a patient. PURPOSE: This study examined the effect of single versus multiple actuations of an MDI into reservoir devices on dose delivery of albuterol, with 3 clinically available reservoir brands. METHODS: An in vitro lung model simulated inspiration from the MDI reservoir system. Albuterol (Proventil; Schering) was delivered by MDI, with the Monaghan Aerochamber, the Diemolding Healthcare Division (DHD) aerosol cloud enhancer (ACE), and the Schering InspirEase, using standardized volumes and inspiratory flows of 30 L·min^{-1}. The MDI was actuated into each brand of reservoir 1, 2, or 3 times in rapid succession, followed by a single inhalation. Aerosol dose at the reservoir mouthpiece was captured on a cotton filter, dissolved in ethanol, and measured with a spectrophotometer at 278 nm. RESULTS: For all 3 brands of reservoir, less accumulated dose of drug is delivered with multiple actuations than with multiple single actuations each followed by inhalation. The total dose in milligrams increased significantly with 2 multiple actuations compared with 1 actuation in the aerochamber and ACE ($p < 0.01$), but not in the InspirEase ($p > 0.05$). The Aerochamber, ACE, and InspirEase delivered a mean total dose (SD) of 0.0264 mg (0.012), 0.0271 mg (0.007), and 0.0136 mg (0.006), respectively, with 1 actuation compared to 0.0485 mg (0.011), 0.0453 mg (0.013), and 0.0218 mg (0.009) with 2 multiple actuations. The increase in total dose with 3 multiple actuations was not significant compared to 2 actuations for any of the brands tested ($p > 0.05$). Although total dose increased with multiple actuations, a decline in efficiency was seen with 2 and 3 multiple actuations, compared to single actuation. The dose delivered per actuation decreased for the Aerochamber, ACE, and InspirEase from 0.0264 mg (0.012), 0.0271 mg (0.007), and 0.0136 mg (0.006) with 1 actuation, to 0.0243 mg (0.006), 0.0226 mg (0.006), and 0.0109 mg (0.005), respectively, with 2 multiple actuations, for losses of 8.0%, 16.6%, and 19.9% in dose per actuation for each brand. A further decline in delivery per actuation to 0.0164 mg (0.001), 0.0184 mg (0.004), and 0.0097 mg (0.005) for the 3 brands, respectively, was found with 3 multiple actuations before inhalation. This was a loss of 37.9%, 32.1%, and 28.7% of the dose per single actuation in each brand. There was no significant difference between the Aerochamber and the ACE in dose availability with 1, 2, or 3 actuations, but both of these brands provided significantly more drug than the InspirEase. CONCLUSION: Maximal aerosol bronchodilator from an MDI reservoir was given by single actuations each followed by a breath. Two rapid actuations followed by a breath will give a significant accumulation of dose with some loss when compared to 2 single actuations each followed by inhalation. Three multiple actuations led to a loss of approximately one third of the drug dose obtainable with 3 single actuations each followed by inhalation, for all 3 brands.

Ventilator-Associated Pneumonia Due to *Pseudomonas Aeruginosa*—SC Brewer, RG Wunderink, CB Jones, KV Leeper Jr. Chest 1996;109 (4):1019.

OBJECTIVE: Ventilator-associated pneumonia (VAP) caused by *Pseudomonas aeruginosa* has been associated with higher case fatality rates than VAP caused by other bacterial etiologies. The causes of this excess mortality are unclear. DESIGN: Retrospective review of 38 consecutive ventilated patients with Pseudomonas pneumonia, documented by highly reliable methods. Charts of 5 additional patients were unavailable for review. SETTING: Medical ICUs of a university-affiliated Veterans Affairs Medical Center and a university-affiliated municipal hospital. MEASUREMENTS: Prospectively collected hospital admission acute physiologic and chronic health examination (APACHE) II scores and cause of ICU admission. Retrospectively calculated organ failure and APACHE scores, VAP score. Clinical and microbiologic variables. Antibiotic treatment and outcome. Direct cause of death by standard definitions. RESULTS: Overall mortality was 69% (26/38), significantly higher than the APACHE II predicted mortality of 42.6% ($p = 0.037$). At least 38% (10/26) of deaths were directly attributable to Pseudomonas VAP. Multivariate analysis of factors associated with death found infectious cause for ICU admission (odds ratio [OR] = 8.67; 95% confidence interval [CI], 0.86 to 85.94) and number of organ dysfunctions on the day of diagnosis (OR = 1.73, 95% CI, 1.02 to 2.92) were significant. Septic shock from Pseudomonas VAP, septic shock from subsequent infection, and multiple organ dysfunction syndrome were the most common immediate causes of death. Mortality increased linearly with increasing APACHE II score on the day of diagnosis. Of initial antibiotic regimens, 67% (26/36) were considered failures. Persistent pneumonia occurred in 35% of patients while recurrent pneumonia was unusual (1/38). CONCLUSIONS: Development of Pseudomonas pneumonia results in a mortality rate in excess of that due to the presenting illness. The attributable mortality determined by several means appears to approach 40%. The excess mortality appears to be related to the host defense response to the pneumonia rather than any characteristic of the pneumonia. Even standard antibiotic regimens fail frequently and do not prevent the excess mortality. Since at least 38% of deaths can be directly attributable to the Pseudomonas pneumonia, improvement in therapy is needed.

The Effects of Neuromuscular Paralysis on Systemic and Splanchnic Oxygen Utilization in Mechanically Ventilated Patients—PE Marik, D Kaufman. Chest 1996;109(4):1038.

OBJECTIVE: To evaluate the effect of neuromuscular paralysis on systemic and splanchnic oxygen utilization in patients in respiratory failure during controlled mechanical ventilation. SETTING: A university-affiliated teaching hospital. INTERVENTION: Mechanically ventilated patients, who were undergoing hemodynamic monitoring and who had a gastric intramucosal pH (pHi) of less than 7.35, were studied. Prior to paralysis, the patients were sedated with lorazepam and morphine to standard end points, and the cardiac output and oxygenation were optimized. The patients were then paralyzed with doxacurium and the ventilator rate adjusted to keep the P_{aCO_2} at baseline value. The hemodynamic and oxygenation profile and pHi were determined prior to paralysis and repeated 2 to 2.5 hours later. RESULTS: Eight patients were studied; their mean age was 63 ± 8 years and acute physiology and chronic health evaluation II score was 22 ± 4. The mean fraction of inspired oxygen, positive end-expiratory pressure, and venous admixture ratio prior to the study was 0.7 ± 0.14, 11.8 ± 2.4 cm H_2O, and 26 ± 9%, respectively. Prior to paralysis, the mean set assist controlled ventilation rate was 15 ± 2 breaths/min and the patient rate was 23 ± 5 breaths/min. With neuromuscular paralysis, the cardiac index fell from 4.6 ± 2.2 to 4.3 ± 2.4 L/min/m^2 ($p = 0.1$), the oxygen delivery fell from 537 ± 129 to 471 ± 95 mL/min/m^2 ($p = 0.03$), and the oxygen consumption and extraction ratio fell from 200 ± 77 to 149 ± 35 mL/min/m^2 ($p = 0.03$) and 36 ± 5 to 31 ± 10, respectively ($p = 0.2$). The pHi increased from 7.21 ± 0.16 to 7.29 ± 0.1 ($p = 0.02$). CONCLUSION: In critically ill patients in respiratory failure, neuromuscular paralysis decreases whole body oxygen consumption and

increases pH_I. Presumably, by eliminating the work of breathing, there is a redistribution of blood flow from the respiratory muscles to the splanchnic and other nonvital vascular beds.

Cumulative Dose-Response Study of Non-CFC Propellant HFA 134a Salbutamol Sulfate Metered Dose Inhaler in Patients with Asthma—EC Kleerup, DP Tashkin, AC Cline, BP Ekholm. Chest 1996;109:702.

OBJECTIVE: This study compares the safety and efficacy of HFA 134a salbutamol sulfate (Airomir in the 3M CFC-free system [3M Pharmaceuticals]) and CFC 11/12 salbutamol (Ventolin [Allen & Hanburys]) in a cumulative dose-response (1, 1, 2, 4, 8 inhalations at 30-min intervals) study in asthmatic patients. DESIGN: Randomized, single-blind, 2-period crossover study. PARTICIPANTS: Twenty-four stable mild to moderate asthmatics. MEASUREMENTS & RESULTS: At all cumulative inhalations, the changes in FEV_1 (absolute, percent, and percent predicted) and FVC were equivalent. There was also no significant difference in heart rate, serum potassium level, BP, 12-lead ECG, Holter monitor recordings, or adverse events. Both HFA 134a salbutamol sulfate and CFC 11/12 salbutamol displayed a significant dose-response for FEV_1, $FEF_{25-75\%}$, FVC, serum potassium, heart rate, and systolic BP. CONCLUSIONS: HFA 134a salbutamol sulfate and CFC 11/12 salbutamol produced clinically and statistically similar airway responses and side effects. These results indicate that HFA 134a salbutamol sulfate would be a safe and effective substitute for CFC 11/12 salbutamol.

Effect of Treatment with Zileuton, a 5-Lipoxygenase Inhibitor, in Patients with Asthma: A Randomized Controlled Trial—E Israel, J Cohn, L Dubé, JM Drazen, for the Zileuton Clinical Trial Group. JAMA 1996;275(12):931.

OBJECTIVE: To study the effect of 3 months of treatment with zileuton, an inhibitor of the enzymatic pathway (5-lipoxygenase) leading to leukotriene formation, on disease control in patients with mild to moderate asthma. DESIGN: Randomized, double-blind, parallel-group study in 401 patients. A 10-day placebo lead-in was followed by a double-blind treatment period of 13 weeks. SETTING: Asthma study clinics in university hospitals and private practices. PATIENTS OR OTHER PARTICIPANTS: Patients with mild to moderate asthma (forced expiratory volume in the first second [FEV_1], 40% to 80% of predicted) whose only treatment was inhaled β-agonists. INTERVENTIONS: Treatment with 600 mg or 400 mg of zileuton or placebo (each taken 4 times daily). MAIN OUTCOME MEASURES: Frequency of asthma exacerbation requiring treatment with corticosteroids, use of inhaled β-agonists, pulmonary function tests, asthma symptom assessment, and quality-of-life evaluation. Safety was evaluated by monitoring adverse events. RESULTS: Only eight (6.1%) of 132 patients receiving 600 mg of zileuton 4 times a day required corticosteroid treatment for asthma versus 21 (15.6%) of 135 patients receiving placebo ($p = 0.02$), giving a relative risk of 2.6. At the time of expected peak drug concentration, the average FEV_1 improved 15.7% in the 600-mg zileuton group versus 7.7% in the placebo group ($p = 0.006$). Quality-of-life assessments significantly improved in the 600-mg zileuton group and not in the placebo group ($p = 0.007$ for the overall score). Elevations in liver function tests (more than 3 times normal), all of which reversed with drug withdrawal, occurred in 5 patients ($p = 0.03$ vs placebo), 3 patients ($p = 0.12$ vs placebo), and no patients treated with 600 mg of zileuton, 400 mg of zileuton, or placebo, respectively. CONCLUSIONS: Three months of 5-lipoxygenase inhibition produced a significant improvement in asthma control. These data indicate that 5-lipoxygenase products of arachidonic acid metabolism are mediators of inflammation with an important role in the biology of asthma.

Adenosine Infusion Improves Oxygenation in Term Infants with Respiratory Failure—GG Konduri, DC Garcia, NJ Kazzi, S Shankaran. Pediatrics 1996;97(3):295.

OBJECTIVE: Adenosine infusion causes selective pulmonary vasodilation in fetal and neonatal lambs with pulmonary hypertension. We investigated the effects of a continuous infusion of adenosine on oxygenation in term infants with persistent pulmonary hypertension of newborn (PPHN). DESIGN: A randomized, placebo-controlled, masked trial comparing the efficacy of intravenous infusion of adenosine to normal saline infusion over a 24-hour period. SETTING: Inborn and outborn level III neonatal intensive care units at a university medical center. PARTICIPANTS: Eighteen term infants with PPHN and arterial postductal PO_2 of 60 to 100 torr on inspired O_2 concentration of 100% and optimal hyperventilation ($P_{aCO_2} < 30$ torr) were enrolled into the study. Study infants were randomly assigned to receive a placebo infusion of normal saline, or adenosine infusion in doses of 25 to 50 μg/kg/min over a 24-hour period. RESULTS: Nine infants each received adenosine or placebo. The 2 groups did not differ in birthweight, gestational age, or blood gases and ventilator requirements at the time of entry into the study. Four of 9 infants in the adenosine group and none of the placebo group had a significant improvement in oxygenation, defined as an increase in postductal P_{aO_2} of ≥ 20 torr from preinfusion baseline. The mean P_{aO_2} in the adenosine group increased from 69 ± 19 at baseline to 94 ± 15 during 50 μg/kg/min infusion rate of adenosine and did not change significantly in the placebo group. Arterial blood pressure and heart rate did not change during the study in either group. The need for extracorporeal membrane oxygenation, incidence of bronchopulmonary dysplasia, and mortality were not different in the 2 groups. CONCLUSION: Data from this pilot study indicate that adenosine infusion at a dose of 50 μg/kg/min improves P_{aO_2} in infants with PPHN without causing hypotension or tachycardia. Larger trials are needed to determine its effects on mortality and/or need for extracorporeal membrane oxygenation in infants with PPHN.

Improvement in Pulmonary Function and Elastic Recoil after Lung-Reduction Surgery for Diffuse Emphysema—FC Sciurba, RM Rogers, RJ Keenan, WA Slivka, J Gorcsan III, PF Ferson, JM Holbert, ML Brown, RJ Landreneau. N Engl J Med 1996;334(17):1095.

BACKGROUND: Pulmonary function may improve after surgical resection of the most severely affected lung tissue (lung-reduction surgery) in patients with diffuse emphysema. The basic mechanisms responsible for the improvement, however, are not known. METHODS: We studied 20 patients with diffuse emphysema before and at least 3 months after either a unilateral or a bilateral lung-reduction procedure. Clinical benefit was assessed by measurement of the 6-minute walking distance and the transitional-dyspnea index, which is a subjective rating of the change from base line in functional impairment and the threshold for effort- and task-dependent dyspnea. Pressure-volume relations in the lungs were measured with static expiratory esophageal-balloon techniques, and right ventricular systolic function was assessed by echocardiography. RESULTS: The patients had significant improvement in the transitional-dyspnea index after surgery ($p < 0.001$). The mean ($\pm$ SD) coefficient of retraction, an indicator of elastic recoil of the lung, improved (from 1.3 ± 0.6 cm of water per liter before surgery to 1.8 ± 0.8 after, $p < 0.001$). Sixteen patients with increased elastic recoil had a greater increase in the distance walked in 6 minutes than the other 4 patients, in whom recoil did not increase ($p = 0.02$). The improved lung recoil led to disproportionate decreases in residual volume as compared with total lung capacity (16% vs 6%), but the decreases in both values were significant ($p < 0.001$). Forced expiratory volume in 1 second increased (from 0.87 ± 0.36 to 1.11 ± 0.45 liters, $p < 0.001$). End-expiratory esophageal pressure also decreased ($p = 0.002$). These improvements in lung mechanics led to a decrease in the partial pressure of arterial carbon dioxide from 42 $\pm$ 6 to 38 $\pm$ 5 mm Hg ($p = 0.006$). Furthermore, the fractional change in right ventricular area, an indicator of systolic function, increased from 0.33 ± 0.11 to 0.38 ± 0.10 ($p = 0.02$). CONCLUSIONS: Lung-reduction surgery can increase the elastic recoil of the lung in patients with diffuse emphysema, leading to short-term improvement in dyspnea and exercise tolerance. *See the related editorial:* ***Surgery To Reduce Lung Volume****—DR Dantzker, SM Scharf. N Engl J Med 1996;334(17):1128-1129.*

What We Say versus What We Do

I'm sure that it's not exactly 'hot' news that the health care industry is in a state of incredible change.[1] While we acknowledge countless examples of different approaches to re-engineering, restructuring, 'downsizing,' vertical integration, horizontal integration, and on and on and on, we must never forget that the prime catalyst is the need for our health-care delivery system to make the transition from a cost-plus or fee-for-service payment system to a capitated or risk-related payment system. If such a transition were not called for as part of an effort to control health-care expenditures, then I feel it's safe to assume that restructuring, re-engineering, and downsizing would be no more popular in the 90s than it was in earlier decades.

Many health-care providers have witnessed elimination of their jobs. No category of health-care worker from CEO on down (even including chaplains) has failed to experience some degree of job elimination in acute care settings. Certainly, respiratory care practitioners are no exception.

Our profession has resisted efforts to eliminate jobs because we believe that we are best able to render respiratory care procedures. Many of us believe that restructuring and re-engineering are the major culprits driving job elimination—but this simply isn't true. We often draw the comparison between disease and symptoms, and I think it's appropriate to recognize the 'disease' as an ever-decreasing utilization of services offered by hospitals. The symptoms of the disease are downsizing, restructuring, and re-engineering. Put another way, if hospital utilization continues to decrease, jobs of all types will be eliminated. Even if there were no efforts to restructure and re-engineer, put simply it's a matter of supply and demand. As demand for services decreases—if nothing else changes—then it follows that fewer human resources will be needed to meet the reduced demand.[2] This will happen with or without restructuring or re-engineering.

I'm sure that many of you realize the true dynamics of change in health care. But what we seem to be struggling with, in some instances, is a difference between what we say and what we do. I believe that one particular activity needs immediate attention because many of the conceptions regarding respiratory care practitioners and what we do seem to flow from this activity. Let's call it the treat-and-leave scenario. Have you ever watched a respiratory care practitioner enter a patient's room, start the patient on a small-volume nebulizer treatment and then leave the room, perhaps to start another treatment? Have you ever watched one of your colleagues initiate an aerosol treatment, remain in the room (physically), but seem to notice everything in the room except the patient? I'm sure this is a rarity throughout the United States. The reason I know it must be flows from the fact that we as a profession consistently argue against downsizing. We consistently argue against transferring so-called routine respiratory care treatments to non-respiratory care practitioners, and we support both arguments by saying that we are the only profession trained and qualified to render respiratory care procedures. We know how to operate the equipment, instruct the patient, coach the patient, and constantly evaluate and assess the patient on behalf of the attending physician. We then convey our findings to the attending physician through appropriate communication channels. We also aggressively seek order changes when, in our opinion, after performing appropriate assessment, we believe that the current order does not match the patient's needs. We thereby influence use of services, in addition to rendering the care itself, and we most certainly have strong communication ties with nurses and other members of the multidisciplinary team in order to keep them abreast of the patient's progress from the perspective of the respiratory care practitioner.

That's what we like to say,[3-5] but every time a respiratory care practitioner starts a treatment and leaves the room, he or she is saying, "You don't need us to perform this task." Every time a respiratory care practitioner stands idly by the patient's bedside without relating to that patient other than to take an occasional pulse and consult the clock, we're telling all who 'buzz' in and out of that room that they don't need us. Do you ever wonder why some hospital executives and nurses and management consultants believe that respiratory care practitioners are not needed for routine services? It's because they have observed such behaviors in respiratory care practitioners, and they believe what the behaviors are saying rather than what the respiratory care profession is saying in terms of their being indispensable to successful respiratory care interventions.

A few hospitals have effectively transferred responsibility for routine respiratory care procedures to non-respiratory care practitioners. Those non-respiratory care practitioners include registered nurses, licensed practical nurses, unlicensed assistive personnel, and others. To my knowledge, the training provided to these substitute caregivers is marginal at best. Training seems to encompass the motor skills necessary to

render routine treatments and to provide information about operation of the devices. Very little, if any, training addresses the reasons that patients are receiving treatment, appropriate assessment, the role of coaching to optimize therapeutic results, and the ongoing evaluation of the appropriateness of the procedures in light of the patient's condition. Ultimately, these efforts will fall short of the mark in terms of decreasing costs, if for no other reason than the absence of adequate utilization control. Bear in mind that one of the 'assumptions of convenience' that is at work in this scenario is that all orders for respiratory care services are appropriate and remain appropriate for the duration of the order. Now we all know this simply isn't true. Indeed, in some institutions efforts have been made to measure the percentage of inappropriate orders.[6] Yet, when we start a treatment and leave the room or when we fail to interact constantly with the patient throughout the therapy, we are saying that meaningful assessment, coaching, and continual evaluation of the appropriateness of the order are not important. We, therefore, make it easy for some 'downsizer' to come in, identify routine respiratory services as clinical responsibility that can be shifted inexpensively, and have such a recommendation readily accepted.

Many of our colleagues recognize the need to provide treatments correctly. They want to instruct, they want to coach, they want to assess, they want to evaluate, and they want to make recommendations concerning appropriateness of respiratory care orders related to patient condition, but they are 'under the gun.' Many top decision makers in hospitals have conveyed by edict that staffing be fixed at a certain level. In many instances, staffing levels do not permit respiratory care practitioners to render all treatments in the hospital on a one-on-one basis. I have the feeling that this trend is growing, and if it is, perhaps now is a good time for us to move toward creating a system that allows us to 'downshift' certain patients within the hospital. I propose that we develop a system that relegates patients requiring respiratory care treatments to one of two categories—Category A: those who require the services of a respiratory care practitioner; and Category B: those who, after appropriate instruction from a respiratory care practitioner,[4,5,7-9] can self-administer their therapy. I believe that it's better that we formally recognize that many patients receiving respiratory care treatments are in effect—by default—self-administering. (What would you call it when someone starts a treatment and leaves the room?) Why don't we recognize this fact? After all, the days of cost-plus reimbursement are ending. Why not redesign our delivery systems to be compatible with the new system?

Here's how it would work. When patients are initially ordered on respiratory care treatments, a qualified respiratory care practitioner visits the patient and performs an in-depth assessment. This assessment not only addresses the more obvious physical and disease-related aspects of the patient's condition but also seeks out information concerning chronicity and the potential for the patient to require therapy after discharge. Patients who have chronic pulmonary diseases or conditions and who self-administer therapy at home are ideal candidates for such a self-administration program. Once the care team agrees that a particular patient should and could self-administer treatment, then a program of instruction is undertaken by the respiratory care practitioner. During the instruction, the patient's ability to self-administer treatment is assessed, with both physical and psychological factors considered. Once the patient has 'graduated' from the instruction phase, then responsibility for physician-ordered compliance is shared by the patient, the nurse, and the respiratory care practitioner assigned to the patient. The patient, of course, must undertake the treatment consistent with physician orders. The nurse (because she is the person in contact with the patient for the greatest number of hours) can offer reminders and make the respiratory care practitioner aware of noncompliance with physician orders. The respiratory care practitioner monitors the patient's self-treatment, offers tips regarding technique, and, of course, performs daily assessments of the patient's condition and the appropriateness of the respiratory care order. This information plus whatever suggestions and recommendations may be warranted is fed back to the ordering physician.

Of course, the patient in Category A will be treated in a more traditional fashion, utilizing the full range of services of respiratory care practitioners who have enough time to instruct the patient concerning the procedure, coach the patient during the procedure, and evaluate the patient's progress and condition on behalf of the ordering physician. These interventions, coupled with a well-trained respiratory care practitioner who is committed to making a positive difference with each and every patient he or she encounters, will go a long way toward alleviation of the disparity between what some members of our profession say and what they do. Such an approach must recognize 'up front' and honestly that not all patients under today's condition can expect to receive services in some hospitals from respiratory care practitioners. Shuffling tasks to others who are not prepared by education, training, and competency testing is not the answer, and treating and leaving is most certainly not the answer. But, by using our knowledge and recognizing that not every patient needs a respiratory care practitioner every time a treatment is needed, we can move forward as part of the change process in health-care delivery today—rather than continuing to sometimes send a message that has and can continue to hurt patients, nurses, physicians, hospital administrators, third-party payers, and respiratory care practitioners. I believe that we do have an opportunity to make an important difference, without compromising the quality of care. What do you think?

Sam P Giordano MBA RRT
Executive Director
American Association for Respiratory Care
Dallas, Texas

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Reprints: Sam P Giordano MBA RRT, Executive Director, American Association for Respiratory Care, 11030 Ables Lane, Dallas TX 75206.

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Low-Dose Theophylline Therapy—or Sometimes Less Is More

Very provocative results from a study of asthma therapy appear in this issue of RESPIRATORY CARE. Emad[1] performed a 6-month study of 120 asthmatic subjects in 3 groups—receiving standard therapy, standard therapy plus placebo, or standard therapy plus theophylline. The outcome variables were peak expiratory flow (PEFR), forced expiratory volume in 1 second (FEV_1), and forced vital capacity (FVC) to assess lung function and hospital admissions prior to, during, and after the 6-month trial to assess a clinically important effect.

The data show that only in the low-dose theophylline group was there an improvement in lung function. In addition only in this low-dose theophylline group were the hospital admissions lower in the 6 months after treatment. Few side affects were noted. The author concludes "that this mode of treatment could be beneficial with no major or significant side effects." The reason they conclude this is that theophylline given at 250 mg every other day would achieve blood levels well below the levels normally associated with side effects. What makes this study intriguing is the fact that such a low dose of theophylline would have any effect at all.

As with almost any clinical study, this one suffers from certain deficiencies and shortcomings. However, before I dwell on these problems, I need to put a couple of things straight. First, my own experience tells me that clinical investigation can be challenging—even under the best of circumstances. Numerous hurdles have to be overcome to perform even the simplest clinical investigation; as an example, consider the problems with sending a protocol through the Institutional Review Board. Second, additional problems arise when, as in this study, larger numbers ($n > 12$) and/or longer (> 1 week) periods of time are involved. Furthermore, my own experience while in Canada and discussions with my international colleagues convince me that conducting research in other countries adds another degree of difficulty to the enterprise. Accordingly, it is reasonable to suppose that conducting research in a country such as Iran must have presented some formidable challenges to the investigator. Lastly, there is the danger that we may judge these data looking through glasses that are colored by our opinions of the politics of government of this particular country. Science must be above governmental politics. We must take the colored glasses off! With these thoughts in mind and given the perceived degrees of difficulty, let us allow some latitude in assessing this study.

Then, too, in discussing the shortcomings of this study consider many of these comments as an impetus to repeat the study. Because, as we will see, the results of this study are potentially important enough to suggest that they be confirmed.

The details of lung function measurement, especially quality control of the data, I found to be sketchy. This may not be serious as the study does have a number of controls. I would quibble with the statement that the asthma of the patients was moderate given that the group on the whole had an FEV_1 of 50% of predicted. This is important because the effect of drugs used in asthma often is influenced by disease severity. I am also surprised by the homogeneity of the lung function among groups, given the manner of group assignment. It would have been helpful to have had symptoms, peak flows, and AM-PM lability data recorded in patient diaries. Again, diary and global assessments would certainly be easy to add in a follow-up study. It is unclear how the hospital admissions were in fact determined. Then too, the finding that, following the end of theophylline treatment, admissions only decreased after the treatment period seems strange as others have reported clinical deterioration.[2] One would have guessed that admissions would decrease only *during* the treatment period. An explanation for these data would be some sort of beneficial 'rebound' phenomenon, but I do not recall any other study reporting such a phenomenon. The mainstream asthma investigator would have obtained lavage data or even had biopsies performed, but those procedures may not have been feasible at the author's institution. Current practice in asthma studies is to include a defined run-in and wash-out period. A cross-over design would also be indicated. However, there is something to be said for keeping it simple.

There are several laudable features of this study as well as important findings. First, is the number of subjects that were used. This sample size is large enough to have seen some small effects and large enough to be representative of the true population effects. Second, there is an inclusion of a second control group (no placebo tablet). Positive placebo effects are common observations in asthma therapy trials and may have caused the trend toward the reductions in admissions observed in Group 2 (placebo) but not Group 1 (standard therapy).

Now let's turn to the conclusion of this study; the important findings are three. First, that a drug given well below the commonly prescribed dose of 10 mg/kg/24 h would show such important effects, is very intriguing. The role of theophylline in asthma treatment is unclear, and its mechanism of action is an enigma.[3,4] At one time it was widely used in North America and was commonly thought to work as a relaxant of

bronchial smooth muscle—a bronchodilator.[4] But we know from numerous clinical studies that theophylline is, in fact, a poor bronchodilator. Theophylline has also fallen from grace because of, (1) reports of severe reactions, (2) need to monitor serum levels, (3) potential drug interaction, and (4) 'fussy' pharmacodynamics.[3,4] In an attempt to rehabilitate this drug within the new order of asthma therapy and inflammation theories,[5] recent studies have focused on anti-inflammatory properties of this drug.[6-8] As a result the best explanation for the current data is that at low doses theophylline must have some unidentified profound anti-inflammatory effect. This finding must be further explored, if for no other reason, because theophylline is a relatively cheap medication that is delivered by what patients like best—a pill.[9]

The finding that theophylline at such a low dose would be effective as found in the current study is truly startling. There are other studies of low-dose theophylline.[6,10] Sullivan et al[6] treated 19 subjects with 200 mg bid which is clearly higher than that of 250 every other day of the current study. They found a reduced number of eosinophils in the airways biopsy and a reduced bronchial constriction in response to antigen challenge. What is not clear in the Sullivan study or in the current study by Emad is what rationale was used to decide on these dosages. Nevertheless, these studies both suggest that we revisit the efficiency and dosage schemes used for this particular drug.

The second important finding was the size of the lung function response and the reduced number of hospitalizations in the treatment group. Most asthma drugs go into further development because they improve patient perception of asthma symptoms—Not a bad approach, but potentially misleading and dangerous given the known problem some patients have in detecting acute airflow limitations.[10] But, the current study shows a 500-mL improvement in FEV_1 (58 to 68% of predicted and a PEFR increase of 1.2 L/s, or 72 L/min), clearly a clinically important improvement. The reduced hospitalization is important by any estimation. This is especially so if one considers, as we must these days, the cost of such untoward events.

The mechanism of action of many drugs that we use is unclear. We use them because we 'know' they work, but how much better could we use them if we understood their mechanism of action? In my mind that is the important 'take-home' message of the current study. Because, if the data are correct, a new and better treatment for asthma will result by merely changing a simple thing—the dose. A lot is being made of the poor compliance of patients in taking their asthma medication.[9] One provocative conclusion might be that poor patient compliance may be a consequence of patients' discovering on their own that lower doses work better.

Charles G Irvin PhD
Professor
Departments of Medicine and Pediatrics
University of Colorado
Health Sciences Center

Director, Pulmonary Physiology Unit
National Jewish Center for Immunology
& Respiratory Medicine
Denver, Colorado

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The Cuff-Leak Test as a Predictor of Postextubation Stridor: A Prospective Study

Paul E Marik MD

BACKGROUND: Postextubation stridor as a result of laryngeal edema has been reported in up to 5% of patients who have received mechanical ventilation for longer than 24 hours. The cuff-leak test has been proposed as a method of determining whether patients can be extubated without developing upper airway obstruction. This test, however, has not been prospectively studied. METHODS: 100 consecutive patients who had been intubated for longer than 24 hours were studied prior to extubation. The cuff-leak test was performed by two different methods. The endotracheal cuff was totally deflated, the lumen of the endotracheal tube was then occluded, and the presence (small or large) or absence of a peritubular leak during spontaneous ventilation was determined. The test was then repeated with positive pressure ventilation using the patients' pre-weaning tidal volume. The results were not available to the intensivist, and the patients were extubated regardless of the results of the leak tests. RESULTS: The mean (SD) length of intubation was 3.8 (3.2) days. Six patients had no leak with both tests, 22 patients had a small leak with both tests, and 60 patients had a large air leak with both tests. Twelve patients had a small leak with one test and a large leak with the other. Two patients developed postextubation stridor. Both were successfully treated with aerosolized racemic epinephrine. These two patients had no leak with either test, making the likelihood of developing stridor significantly higher in the group of patients that had no peritubular leak prior to extubation ($p = 0.003$). The cuff-leak tests had a negative predictive value of 100% but a positive predictive value of only 33%. CONCLUSION: The presence of a cuff leak indicates that postextubation stridor is unlikely. Although the absence of a cuff leak does not guarantee extubation difficulty, such patients should be observed closely during the immediate postextubation period [Respir Care 1996;41(6):509-511].

Introduction

Stridor is a relatively uncommon complication in adults who have been extubated. However, it may be associated with serious complications and can lead to death.[1-4] This complication is believed to be caused by edema of the subglottic portion of the trachea.[2-5] Most patients develop an air leak when the cuff of the endotracheal tube is deflated. It has been suggested that an air leak may be absent in patients with upper-airway edema.[6] The primary aim of this study was to deter-

The author is with the Department of Critical Care Medicine and Respiratory Therapy, St Vincent Hospital, Worcester, Massachusetts.

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Reprints: Dr Paul E. Marik, Department of Critical Care, St Vincent Hospital, 25 Winthrop Street, Worcester MA 01604.

mine the positive and negative predictive value of the cuff-leak test in predicting postextubation stridor. A secondary aim of the study was to determine the incidence of postextubation stridor in a heterogenous group of ICU patients.

Patients & Methods

This study was conducted in the medical and surgical intensive care units (ICUs) at St Vincent Hospital, a University-affiliated teaching hospital in Worcester, Massachusetts. Approval to conduct this study was obtained from our Institutional Review Board who waived the requirement of informed consent. During the study period, 100 consecutive patients who had been intubated for longer than 24 hours were prospectively followed until elective extubation. Cardiothoracic patients were excluded from study. The patients were each included only once in the study. All patients were sedated with lorazepam, either alone or in combination with morphine. Endotracheal tubes were secured using adhesive tape by the method described by Levy and Griego.[7] Cuff pressures were routinely checked every 8 hours, with an accepted limit of 25 cm H_2O.

Prior to extubation, the cuff-leak test was performed by two different methods. (1) The endotracheal cuff was completely deflated, the lumen of the endotracheal tube was occluded, and the presence (small or large) or absence of a peritubular leak during spontaneous ventilation was assessed. (2) The test described in (1) was repeated during positive pressure ventilation using the patients' preweaning tidal volume. The results of the tests were not available to the intensivist, and the patients were extubated regardless of the results of the leak tests.

All patients were observed for the development of signs of upper-airway obstruction (ie, a prolonged inspiratory phase associated with recruitment of accessory respiratory muscles), so-called laryngeal dyspnea,[8] for 24 hours following extubation. Laryngeal edema was diagnosed when either upper-airway obstruction or stridor (defined as a 'crowing' sound during inspiration) occurred.

For each patient, we recorded the patient's age and sex, the primary disease and reason for endotracheal intubation, the APACHE II score on admission to the ICU,[9] the size of the endotracheal tube, the route of intubation, and the duration of intubation. Treatment administered for stridor and the patient's response and the use of systemic corticosteroids prior to extubation were also recorded. The number of patients who required reintubation within 24 hours of extubation was recorded as well as the reasons for reintubation.

At the end of the data collection, summary statistics were compiled to allow a description of the study population. Data are presented as mean (SD) or proportions, as appropriate. The Fisher's exact test (two-tail) was performed to compare the groupwise likelihood of developing stridor if there were no cuff leak (both tests) compared to developing stridor if a cuff leak were present.

Results

One hundred consecutive patients (61 men) were evaluated. Their age and APACHE II scores were 57 (9) years and 14.9 (2.5). Of the 100 patients, 53% were intubated for respiratory failure and 38% required postoperative ventilation. None of the patients had suffered trauma or burns. Twenty-one percent of patients were receiving systemic corticosteroids. Duration of intubation was 3.8 (3.2) days. All but 2 patients were intubated orally. A size 8-mm (internal diameter) tube was used in 58% and a size 7.5-mm tube in 24% of patients.

Six patients had no leak with either test, 22 patients had a small leak with both tests, and 60 patients had a large air leak with both tests. Twelve patients had a small leak with one test and a large leak with the other. Two patients developed postextubation stridor, one of whom developed mild upper-airway obstruction. These two patients had no leak with either test. The first patient was a 67-year-old man who had been intubated for 3 days with a size 7.5-mm tube for the management of pulmonary edema. The second patient was a 68-year-old man who had undergone a pneumonectomy for a pulmonary malignancy. He had been intubated with a size 9-mm, single-lumen endotracheal tube for 36 hours following surgery. Both of these patients were successfully treated with aerosolized racemic epinephrine. The difference in the incidence of stridor between the two groups (with and without a cuff leak) was statistically significant ($p = 0.003$). The cuff-leak tests had a negative predictive value of 100% (percent of patients who did not have stridor, given the presence of a peritubular leak) and a positive predictive value of 33% (percent of patients who developed stridor, given the absence of a peritubular leak). Two patients with chronic obstructive airways disease required reintubation for respiratory distress 4 hours and 9 hours after extubation. Neither of these patients had evidence of laryngeal edema.

Discussion

Laryngeal edema has been reported to occur in 2-15% of patients after tracheal extubation.[1-4,8,10-14] The difference in the rates observed in different studies may be ascribed to differences in the populations studied, the tube material, or the criteria used for the diagnosis of laryngeal edema. Pediatric patients appear particularly vulnerable because of the small airway and the looseness of the tissue in the submucosa.[10,11] In a multicenter, prospective study performed in an adult population, the overall incidence of laryngeal edema postextubation was 4.2% with only 1% of patients requiring reintubation for airway compromise.[14] These authors identified female gender and intubation for longer than 36 hours as the only risk factors predictive of postextubation laryngeal edema. Other authors have implicated traumatic intubations,[3,4,13] the use of large-size tubes,[8,13-15] and overinflated cuffs[5,14] as risk

factors for the development of this complication. The reason for the low incidence of laryngeal edema in our study is unclear. The high percentage of patients on systemic corticosteroids, the relatively short duration of intubation, or the method used for fixation of the endotracheal tube may have contributed to this finding. Furthermore, none of our patients had suffered traumatic injuries or burns, a group of patients at high risk of developing postextubation stridor.[1,2,14] In addition, the use of low-pressure, high-compliance cuffs, improved sterilization techniques, and improved endotracheal-tube care may also have contributed to this result.

Damage from endotracheal tubes to mucosa in the airway is immediate and appears to result from the mechanical effects of the tube. The two major mechanisms that have been postulated to cause laryngeal injury from an endotracheal tube are the movement of the tube with abrasion of the mucosa and pressure necrosis.[2,5] Radiologic examinations have shown that an endotracheal tube moves, on average, 3.8 cm when the head is moved from flexion to extension.[16] Levy and Griego[7] have demonstrated that the method of tube fixation affects the degree of endotracheal tube movement. They found that the adhesive-tape method of fixation, as used in our study, was associated with the least amount of tube movement. The manufactured curve of the endotracheal tube is reversed as it passes over the posterior portion of the cricoid plate, and, because the area of contact is small, the pressure exerted is exceedingly high.[5] In addition, animal experiments have demonstrated that the area of laryngeal ischemia is related to the size of the endotracheal tube.[5]

Fisher and Raper[17] evaluated the value of the cuff-leak test in patients with upper airway problems. In their first cohort of patients, if no leak was detected, the cuff was left deflated and the patients re-evaluated subsequently. All but two patients developed a cuff leak with time and were successfully extubated. Two patients were extubated in the absence of a cuff leak and both required reintubation. The predictive value of the cuff-leak test in this experiment is difficult to interpret. However, these authors also reported on 10 patients who were extubated in the absence of a cuff leak.[17] Of these, 7 were extubated uneventfully, and 3 required reintubation. This latter result supports the findings of our study—a positive predictive value of approximately 33%.

In conclusion, the results of this study suggest that significant upper-airway obstruction is an uncommon complication following short-term endotracheal intubation in a unselected sample of ICU patients and that only patients at high risk of developing this complication require pre-extubation screening. Our data suggest that upper-airway obstruction and/or stridor are unlikely to develop in patients who have an air leak when the endotracheal-tube cuff is deflated. However, a significant proportion of patients who have no leak can also be successfully extubated. These latter patients, however, are at an increased risk of requiring reintubation and should be closely observed during the postextubation period.

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Imposed Work of Breathing during Tracheal Pressure-Triggering Using a Demand-Valve CPAP System*

Shashi B Bhatt MD, Chandra B Patel MD, Ian KS Tan MRCP, Deborah Vernasco MD, and YH Tam BS MPhil

BACKGROUND: Levels of work of breathing imposed (WOB_i) by variations in ventilator design have been addressed by a number of investigators. We conducted this study to assess the effects of tracheal pressure-triggering (TPT) on the WOB_i and to compare it with conventional pressure-triggering (CPT). MATERIALS & METHODS: Sine wave breaths of ≅ 500 mL, with mean flows of 30 and 60 L/min, were inspired through a test ventilator (Servo 900 C) set in the continuous positive airway pressure (CPAP) mode (CPAP, 5 cm H_2O). WOB_i was measured during CPT and TPT. In addition, we simulated 500-mL breaths through the test ventilator with its trigger sensitivity set at −10 cm H_2O for both CPT and TPT. Pressure was measured at the Y-piece and within the tracheal lumen. Flow was measured at the Y-piece and volume obtained from the digital integration of flow. WOB_i was calculated from the area enclosed by the pressure-volume plot (in that part of the pressure-volume loop where the pressure was below the level of PEEP). RESULTS: WOB_i (including that contributed by the tracheal tube) was less during TPT, mean (SD) 258 (42) mJ/L than with CPT, 477 (24) mJ/L at 60 L/min, $p < 0.05$. However, for breaths simulated at flows of 60 L/min, mean inspiratory pressures within the tracheal lumen and at the Y-piece were greater during TPT than during CPT, $p < 0.05$ for both. Under the test conditions used to measure trigger delay and trigger work, trigger delay was similar during TPT and CPT, 280 (20) ms and trigger work was similar for the two triggering modes 1.34 (0.3) cm $H_2O \cdot s$ vs 1.31 (0.25) cm $H_2O \cdot s$. CONCLUSIONS: Decrease in WOB_i during TPT is not due to an alteration in triggering function per se but to an inadvertent pressure assist and is no more advantageous than application of pressure support to compensate for apparatus work. [Respir Care 1996;41(6):512-518]

Dr Bhatt is a Fellow in Critical Care Medicine, Dr Patel and Dr Vernasco are Clinical Assistant Professors of Anesthesiology—Maricopa Medical Center, Phoenix, Arizona. Dr Tan is Lecturer, and Mr Tam is Scientific Officer—Chinese University of Hong Kong, United Kingdom.

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Correspondence: Shashi B Bhatt, Department of Anesthesiology, Maricopa Medical Center, 2601 E Roosevelt, Phoenix AZ 85010.

*See the related Points of View on pages 524-528

Background

When patients in acute respiratory failure begin to recover from the initial precipitating event, they are gradually weaned from mechanical ventilation. During the process of weaning, patients assume a progressively greater proportion of the work necessary to ventilate the lungs. In this phase, the patients must perform work not only to overcome the impedances of their own respiratory system but also the work imposed by the ventilator demand valve, tracheal tube, connectors, humidifiers, and the ventilator circuitry. This imposed work can sometimes become a large fraction of the total

patient work and may contribute to difficulties in weaning patients from mechanical ventilation.[1,2] This has been termed nosocomial respiratory failure or iatrogenic ventilator dependency.[3] Efforts have been directed in recent years toward documenting the imposed work of different ventilators and circuits. Ventilator designers have responded by addressing the ventilator pneumatics and electronics to diminish the response time of demand valves and altering the inspiratory flow algorithm to better respond to patient needs, thereby decreasing the imposed work.

An innovative method for decreasing the imposed work has been suggested by Banner and colleagues.[4] During conventional pressure-triggering, the ventilator pressure transducer that senses the decrease in airway pressure is located within the ventilator, in the exhalation part of the ventilator circuitry. In order to decrease the pressure in this transducer, Banner et al[4] suggested that the patients must overcome the impedances imposed by the tracheal tube, ventilator tubing, and the expiratory flow transducer. They suggested that sensing pressure at the carinal end of the tracheal tube and conveying that signal directly to the sensing transducer (tracheal pressure-triggering) would considerably reduce the imposed work. The concept of tracheal pressure-triggering for demand-valve CPAP systems appears attractive. Tracheal sensing moves the location of the sensing point closer to the contracting respiratory muscles. Moving the sensing point closer to the contracting respiratory musculature (as is also achieved during pleural pressure support[5]) has been considered advantageous. However, in contrast to the low signal-to-noise ratio in pleural pressure measurements, tracheal pressure measurements are more stable and therefore better suited as a trigger source. Using this strategy, Banner and colleagues[4] demonstrated that the imposed work was considerably reduced when compared with conventional pressure-triggering. We undertook bench re-evaluation of the work of Banner and colleagues,[4] prior to clinical application of tracheal pressure-triggering.

Materials & Methods

Spontaneous breathing was simulated using a bench model similar to that used by other investigators.[6,7] The inspiration simulator was constructed from a two-compartment lung model. The two compartments of the lung model are connected by a bar so that when one compartment is mechanically ventilated it actively displaces the other compartment, simulating inspiration. The system was driven by a Hamilton Veolar Ventilator,* using a sine wave pattern of inspiratory flow. Inspiratory time was set at 25% of the total respiratory cycle, and the tidal volume (V_T) was set at 500 mL. The inspiratory flow ($\dot{V}$) of the driving ventilator was altered by varying the respiratory rate (breaths/min, f) to yield mean inspiratory flows of approximately 30 and 60 L/min. The inspiratory time for breaths simulated at 30 L/min was 1 s (f = 12) and for breaths simulated at 60 L/min was 0.5 s (f = 24). The choice of the respiratory rates and inspiratory times was made with due regard for the avoidance of auto-PEEP. The compliance of both the driving and the simulating 'lungs' was set at 50 mL/cm H_2O.

The lung model was in turn connected to the test ventilator (Servo 900 C). The ventilator used in the present study was taken from routine clinical use and was not specially calibrated or serviced for this study. The test ventilator was set in the CPAP mode (CPAP, 5 cm H_2O) with the working pressure set at 60 cm H_2O. To offset the effects of the test conditions due to the increase in 'FRC' in the lung-simulator compartment of the test lung, 5 cm H_2O PEEP was set on the driving ventilator so that both compartments of the lung model would have the same end-expiratory volume. The trigger sensitivity of the support ventilator was set at −2 cm H_2O. No humidifiers were incorporated into the ventilator circuits. Study of the imposed work was conducted with the test ventilator set for conventional pressure-triggering (with the pressure being sensed within the expiratory path of the ventilator). Study of the imposed work was also repeated with the pressure signal measured at the carinal end of the tracheal tube and communicated to the expiratory pressure transducer within the ventilator using a 2-mm inside diameter (ID), noncompliant plastic tubing (tracheal pressure-triggering). Data from 5 consecutive breaths were recorded at each flow and triggering location for subsequent analysis.

The lung simulator was connected to the test ventilator through a model trachea (constructed from 22-mm-ID corrugated tubing) and an 8.0-mm-ID tracheal tube. Embedded within the side wall of the tracheal tube is an additional lumen that opens at the carinal end. This additional lumen was connected to the expiratory pressure transducer of the support ventilator during the tracheal pressure-triggering phase of the study.

$\dot{V}$ was measured using a variable-orifice pneumotachometer. Airway pressures at the Y-piece and within the tracheal lumen were also measured using a Bicore pressure transducer CP-100. The calibration of the pressure and flow transducers was checked against an RT 200 calibration analyzer. The pressure and flow signals were digitized at 50 Hz and stored on an IBM-compatible personal computer (AT 486), using a RS232 communication program. The digitized signals were analyzed in an analog format using a computer program written in Borland C++ by one of the investigators (YHT). Volume was obtained by digital integration of the flow signal. The imposed work of the ventilator in its different triggering modes was estimated by measuring the area enclosed by the airway pressure-volume loop. Only that part of the airway pressure-volume loop, in which the airway pressure was below the level of PEEP, was used for the estimation of the imposed work. The part of the airway pressure-volume loop in which the air-

*Suppliers of commercial products are listed in the Product Sources Section at the end of the text.

way pressure was greater than PEEP was estimated as 'ventilator work' or inspiratory assist (Fig. 1). In the present investigation these areas have not been summed but are expressed separately. The imposed work and the inspiratory assist are expressed as mJ/L.

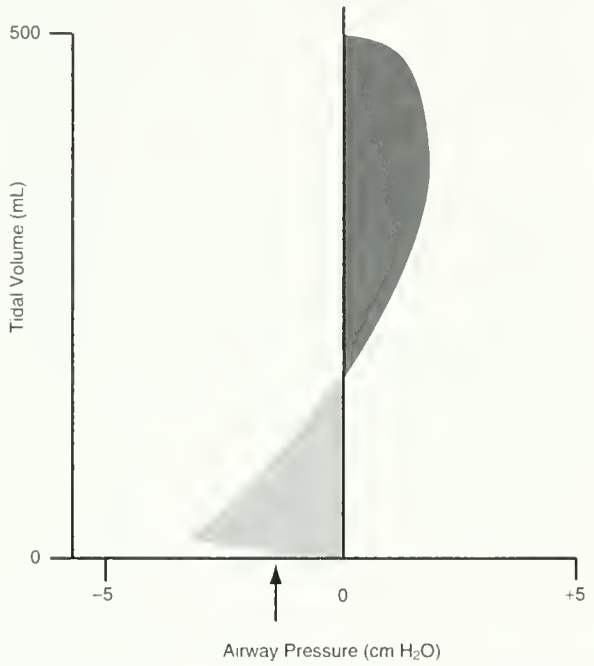

Fig. 1. The inspiratory pressure-volume relationship delineating the inspiratory imposed work and the inspiratory assist. Arrow indicates the point at which the demand valve opens and flow begins. End-expiratory pressure is represented as 0 pressure.

Maximum negative inspiratory pressure was estimated as the lowest pressure below the level of the PEEP. This was estimated from pressures measured both at the Y-piece and within the tracheal lumen.

In order to better delineate the events occurring in the triggering phase of the test ventilator, its trigger sensitivity was set at −10 cm H_2O and a 500-mL breath was inspired through it. Under these test conditions, we measured the trigger delay (time between the first decrement of airway pressure and the onset of airflow) and the trigger work, which was estimated as the pressure-time product between the first decrement of airway pressure (t_0) and the onset of airflow (t_1) (Fig. 2).[8]

$$\text{Trigger work} = \int_{t_0}^{t_1} (\text{PEEP} - P_t)dt,$$

where P = airway pressure; P_t = airway pressure at any instant t.

Comparisons were made between the two triggering modes (conventional pressure-triggering and tracheal pressure-trig-

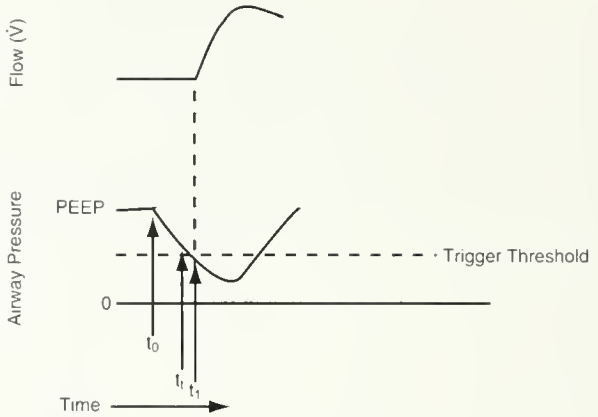

Fig. 2. Estimation of the triggering pressure-time product. t_0 represents the first decrement of airway pressure, t_1 represents the onset of inspiratory airflow and t_t represents the point at which the airway pressure attains the level of the trigger threshold. Triggering pressure-time product is estimated from the integral of the pressure below peak end-expiratory pressure (PEEP) between t_0 and t_1. Time ($t_1 - t_0$) represents the trigger delay. Only a small fraction of the trigger delay is due to the delay in the response of the demand valve ($t_1 - t_t$).

gering) at each flow. Statistical analysis was performed using the student's *t* test for the comparison of the means and a $p < 0.05$ was considered significant. Statistical analysis was performed using SPSS for Windows 6.0. All results are expressed as mean (SD).

Results

The actual V_T measured at the Y-piece of the simulated lung was 517 (10) mL at 30 L/min and 476 (8) mL at 60 L/min, during conventional pressure-triggering. During tracheal pressure-triggering, there appeared to be some augmentation of V_T—550 (12) mL at 30 L/min and 514 (10) mL at 60 L/min $p < 0.05$ at both flows compared with V_T during conventional pressure-triggering.

The imposed work measured for the Servo 900 C ventilator was significantly less during tracheal pressure-triggered breaths, as compared with conventional pressure-triggering both at 30 L/min and at 60 L/min (Table 1). At both flows, the imposed work estimated from pressure measured within the tracheal lumen was greater than that obtained when pressure measured at the Y-piece was used for computation of the pressure-volume plot (Table 1).

The inspiratory assist was measured to be greater for the tracheal pressure-triggered breaths compared to conventional pressure-triggering (Table 1) for breaths simulated at 30 L/min and at 60 L/min.

The mean inspiratory pressure was higher during the tracheal pressure-triggered breaths (measured at the Y-piece and within the tracheal lumen) compared to the conventional pres-

Table 1. Respiratory Variables during Conventional Pressure-Triggering and Tracheal Pressure-Triggering at Flows of 30 and 60 L/min. Pressure and Work Values Are Mean (SD).

Variable	Measurement Site	Conventional Pressure-Triggering	Tracheal Pressure-Triggering	Conventional Pressure-Triggering	Tracheal Pressure-Triggering
		Flow			
		30 L/min	30 L/min	60 L/min	60 L/min
Mean inspiratory pressure (cm H_2O)	Y-Piece	5.56 (0.5)	6.22 (0.1)*	3.09 (0.2)	4.24 (0.2)*
	Trachea	4.84 (0.1)	5.25 (0.1)*	−0.43 (0.15)	1.94 (0.25)*
Maximum negative pressure (cm H_2O)	Y-Piece	−3.0 (0.2)	−0.1 (0.1)*	−7.9 (0.4)	−3.7 (1.2)*
	Trachea	−4.1 (0.2)	−2.1 (0.1)*	−9.2 (0.3)	−6.1 (1.2)*
Inspiratory imposed work (mJ/L)	Y-Piece	38.6 (1.5)	1.8 (0.4)*	98.9 (17)	26.6 (0.9)*
	Trachea	107 (2.6)	13.6 (1.8)*	477 (24)	258 (42)*
Inspiratory assist (mJ/L)	Y-Piece	44.6 (2.2)	272 (18)*	0	592 (22.8)
	Trachea	4.6 (90.68)	173 (10)*	0	34.6 (3.4)*

* $p < 0.05$ compared with conventional pressure-triggering at the same flow.

sure-triggered breaths (Table 1). In fact, during tracheal pressure-triggering the mean inspiratory pressure measured within the tracheal lumen (for a breath with a mean inspiratory flow of 30 L/min) was of the same order of magnitude as the pressure measured at the Y-piece during a conventional pressure-triggered breath (Table 1).

For breaths simulated with the trigger sensitivity set at −10 cm H_2O, the trigger delay was similar for conventional pressure-triggered breaths, compared to the tracheal pressure-triggered breaths, 280 (20) ms. The trigger work estimated under these conditions was also similar during conventional pressure-triggered breaths, compared to tracheal pressure-triggered breaths, 1.34 (0.3) cm $H_2O \cdot s$ versus 1.31 (0.25) cm $H_2O \cdot s$. An X-Y plot of pressure measured at the Y-piece against pressure within the tracheal lumen (from the onset of the first decrement of airway pressure to the point just before the initiation of airflow), yielded a straight line with $r^2 = 0.99$ (Fig. 3).

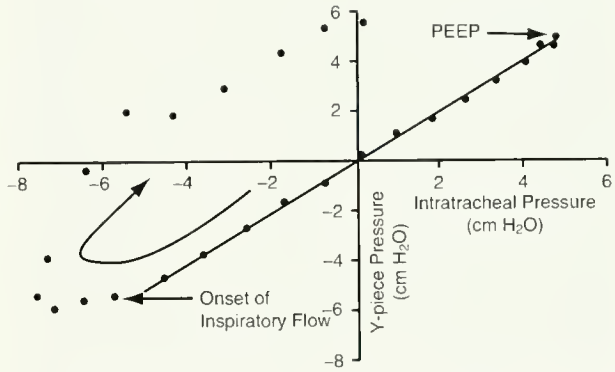

Fig. 3. Intratracheal and Y-piece pressure relationship during the early inspiratory effort, with the trigger sensitivity set at −10 cm H_2O. The breath commences at the point marked peak end-expiratory pressure (PEEP) and then follows the direction marked by the arrow. Both pressures have been recorded at 20-ms intervals. Until the point of initiation of airflow, the intratracheal and Y-piece pressures are identical. After the inspiratory flow begins, the intratracheal pressure is less than the corresponding Y-piece pressure measured at the same time. This difference represents the pressure drop across the tracheal tube (resistance). For tracheal pressure-triggering to reduce trigger delay or trigger work, the rate of decrease of intratracheal pressure must be greater than the rate of decrease of Y-piece pressure prior to the onset of inspiratory flow.

Discussion

The inspiratory imposed work estimated in the present investigation includes the work performed by the patient, not in overcoming the impedances of his own respiratory system (compliance and resistance), but the additional work required to be performed in overcoming the external impedances (tracheal tube, demand valve, ventilatory circuitry).[9]

Numerous investigations of the imposed work of different commercially available mechanical ventilators have been published.[6,7,10-14] However, the results of the present investigation cannot be readily compared with these investigations because the measurement conditions, especially the V_T, $\dot{V}$, and the shape of the inspiratory flow waveforms, are different. Hirsch et al[15] have investigated the imposed work of 9 different commercially available ventilators, using test conditions comparable with those used in the present study. They reported imposed work for a Servo 900 C as 91(30) mJ/L for a 429 mL V_T and a maximum inspiratory $\dot{V}$ of 63 L/min (CPAP 10 cm H_2O). This compares favorably with our determination of the imposed work of 98.9 (17) mJ/L for a 476-mL V_T at a mean flow of 60 L/min. To put these

figures of imposed work in context, the normal inspiratory physiologic work of breathing in healthy adults is approximately 500 mJ/L.[16]

In the present investigation, we have reported the imposed work distinct from the inspiratory assist rather than as a summation of these two (as has been reported by other investigators)[7,10] because we believe that an assist occurring in the later part of inspiration may not completely compensate for the work imposed in the earlier part of inspiration.

Previous studies evaluating devices with differing trigger strategies have used one of two approaches. Either they have measured total patient work in inflating the lung and chest wall or the imposed work (of the extrinsic impedances).[4,17-20] Each strategy has its own advantages and limitations. However, the imposed work includes not only the initial threshold work to trigger the delivery of flow from the ventilator's pneumatic system, but also the work that the patient performs in the face of the early incoming flow when this is less than the patient demand. In the present investigation, the sum of the initial threshold work and this subsequent work has been called the inspiratory imposed work.

The measurement of the imposed work may be important when one is comparing two different devices because of the interest not only in the initial threshold trigger work but also the subsequent sustaining work. However, when one is comparing the same device under two different trigger situations, estimations of the imposed work may not clearly reveal the subtle differences caused by the differences in the trigger work as this may constitute only a fraction of the total imposed work. Only a few investigators have estimated the true triggering work, an estimate of the work expended prior to the opening of the demand valve. Konyukov and colleagues[21] have estimated the triggering work of breathing from the pressure-volume loop of the driving compartment prior to the initiation of gas flow in the test-lung compartment. In the present investigation, we have quantified the triggering work as the pressure-time product prior to the initiation of gas flow.

The main thrust of the present investigation was to assess whether the tracheal pressure-triggering of the demand-valve CPAP system would decrease imposed work, as had previously been suggested,[4] and also to gain insights into the mechanisms of the changes in the imposed work. Our results are in general agreement with Banner et al,[4] in that the apparent imposed work during tracheal pressure-triggering is less than during conventional pressure-triggering. However, the mechanism by which tracheal pressure-triggering reduces the imposed work is unclear at the present time. The early proponents[4] of tracheal pressure-triggering suggested that because the patient would not have to overcome the impedances imposed by the ventilator circuit and the tracheal tube in conveying the pressure decrement of the initial inspiratory effort during tracheal pressure-triggering, the imposed work would decrease. However, the initial part of the decrement of airway pressure that takes place before the opening of the demand valve is unaccompanied by gas flow, and, therefore, the resistance of the system is unimportant.

Two separate mechanisms for apparent reduction in the imposed work may be operative during tracheal pressure-triggering. One of the mechanisms could possibly decrease the initial threshold work or the triggering work by moving the pressure-sensing site closer to the inspiratory force-generating muscles. This mechanism, if effective, would be desirable. The trigger work appears to be composed of two separate components (Fig. 2). The first part of this trigger work is due to the patient's generating the decrement of the airway pressure to the level of the trigger threshold (trigger sensitivity set on the ventilator). This portion of the work is a function of the rate of decrease of airway pressure (dP/dt), which in turn depends on the inspiratory drive and the thoracic volume plus the circuit volume. The second part of the trigger work is caused by the system delay of the ventilator (caused by the electronics and/or pneumatics). If tracheal pressure-triggering is to reduce the triggering work, then the first part of the triggering work would have to be reduced (because it would not cause a change in the system delay). For this to occur the dP/dt of the carinal pressure would have to be greater than the dP/dt of the airway pressure measured at the Y-piece or the exhalation pressure transducer within the ventilator. In the present investigation, we have demonstrated that the dP/dt of the carinal pressure is identical to the dP/dt of the pressure measured at the Y-piece during the triggering phase, prior to the initiation of inspiratory flow (Fig. 3). Moreover, we were unable to demonstrate any difference in the trigger work between the conventional pressure-triggering and the tracheal pressure-triggering for breaths simulated through the ventilator set at a trigger sensitivity of -10 cm H_2O. The trigger delay and trigger work measured with the trigger sensitivity set at -10 cm H_2O in the present investigation are not indicators of the clinical performance of a Servo 900 C ventilator. We made these measurements at a trigger sensitivity of -10 cm H_2O, in order to magnify the events (increase the time between the onset of inspiratory effort and the initiation of gas flow) occurring during the triggering phase. Most of the trigger delay measured in this investigation with the trigger sensitivity at -10 cm H_2O is due to the time taken by the airway pressure to achieve the trigger threshold. Our measure is not meant to be indicative of the inherent system delay of a Servo 900 C because a trigger sensitivity of -10 cm H_2O is unlikely to be used clinically.

A second mechanism that would be effective in decreasing the imposed work would be in operation later in the inspiratory cycle, after the initiation of the inspiratory flow (Fig. 4). During tracheal pressure-triggering, not only does the intratracheal pressure act as a triggering source but it also becomes the sensing/control point for pressure regulation during the subsequent inspiration. During tracheal pressure-triggering, because the pressure control site is downstream of a resistor (tracheal tube), the actual regulated pressure measured

at the Y-piece or within the tracheal tube is greater than the pressures measured at the same sites (Y-piece or within the tracheal tube) during conventional pressure-triggering (Fig. 4). This increased pressure at the Y-piece or within the trachea leads to an inadvertent pressure assist during tracheal pressure-triggering. This inadvertent pressure assist is greater with tracheal tubes of smaller ID (because of the greater pressure drop across the tube at any flow) or higher simulated flows. This is consistent with the results of Banner et al[4] ie, of greater reductions of the imposed work during tracheal pressure-triggering when breaths are simulated through tracheal tubes of smaller ID or at higher inspiratory flows. Evidence that this mechanism of inadvertent pressure assist is making a greater contribution to the decreases in the imposed work during tracheal pressure-triggering comes from two different sources in the present investigation. First, the mean inspiratory pressure is greater during tracheal pressure-triggering as compared with conventional pressure-triggering. Also, there is a larger inspiratory assist during tracheal pressure-triggering as compared with conventional pressure-triggering. Not only is the measured inspiratory assist greater during tracheal pressure-triggering, but we also found a small augmentation of V_T at each flow as compared with conventional pressure-triggering.

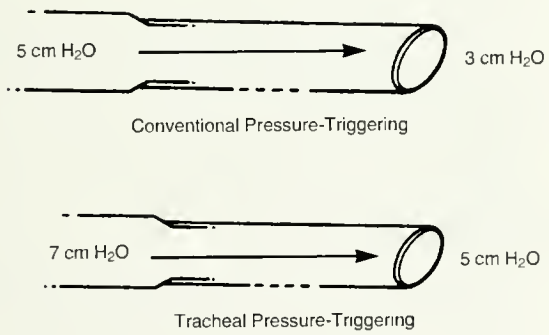

Fig. 4. The mechanism of the inadvertent pressure assist during tracheal pressure-triggering. A hypothetical continuous positive airway pressure system is shown, which regulates the pressure at 5 cm H_2O, both during inspiration and during expiration. The tracheal tube is assumed to represent the principal resistance (4 cm $H_2O \cdot s \cdot L^{-1}$), and the inspiratory flow is assumed to be 30 L/min. During conventional pressure-triggering, the pressure at the Y-piece is 5 cm H_2O, and the 2-cm-H_2O pressure drop across the tracheal tube results in an intratracheal pressure of 3 cm H_2O. During tracheal pressure-triggering, the intratracheal pressure is regulated at 5 cm H_2O, and, therefore, the Y-piece pressure is now 7 cm H_2O, thus resulting in an inadvertent inspiratory pressure assist during tracheal pressure-triggering.

Although tracheal pressure-triggering was found not to affect the trigger delay or the trigger work, it could have one advantage distinct from its effects on the trigger function per se. It has been suggested that low levels of pressure support can be added to overcome apparatus-imposed work.[22] The application of this minimal level of pressure support requires knowledge of tracheal tube ID, the shape of the inspiratory flow waveform, and the inspiratory flow.[22,23] Use of tracheal pressure-triggering can achieve the same effect without the knowledge of these variables. However, this advantage is gained at the expense of additional circuitry that would have to be included in the ventilator. The small lumen of the tube used to convey the pressure signal to the expiratory pressure transducer for achieving tracheal pressure-triggering is vulnerable to blockage by tracheal secretions. So a back-up trigger system would have to be built into the ventilator, adding further costs to the ventilator.

There are limitations to the present study. The pressure and flow signals were digitized at 50 Hz, giving a resolution of 20 ms on the time scale. Hence, smaller differences, if any, between the different triggering modes, would not be readily apparent with the present study. Second, this study has been conducted on a mechanical model, and this may impose limitations when the results are extrapolated to clinical situations. The imposition of a threshold load (induced by the trigger delay) at the commencement of the breath may cause within-breath compensation for these imposed loads,[24] causing a modification of the subsequent portion of the breath. This compensatory effort may modify the actual imposed work. However, this compensatory phenomenon would be absent in the mechanical model tested.

In conclusion, tracheal pressure-triggering does not cause a decrease in the trigger delay or the trigger work. And, although tracheal pressure-triggering was found to decrease the imposed work, this decrease was caused by the ventilator's attempting to regulate the pressure at the carinal end of the tracheal tube thus effecting a small inadvertent pressure assist. We believe that tracheal pressure-triggering confers no more advantage than that obtained by adding pressure support to compensate for the apparatus work.

ACKNOWLEDGMENTS

We thank Arthur Pincus, Biomedical Specialist, for his technical assistance in the conduct of this study and Warren G Sanborn PhD for his review of the manuscript and his many helpful comments and suggestions. The Bicore monitor used in this study was loaned by Bicore Monitoring Systems.

PRODUCT SOURCES

Test Lung:
Vent Aid TTL, Michigan Instruments, Grand Rapids MI

Ventilators:
Hamilton Veolar Ventilator, Hamilton Medical Inc, Reno NV
Servo 900C, Siemens-Elema, Solna, Sweden

Endotracheal Tube:
Hi-LO Jet orotracheal tube, NCC, Mallinckrodt, Argyle NY

Monitoring & Calibrating Equipment:

VarFlex, CP-100, Bicore Monitoring Systems, Irvine CA
CP-100, Bicore Monitoring Systems, Irvine CA
RT 200 Timeter Corp, Lancaster PA

Computer & Software:

IBM-compatible personal computer (AT 486)
SPSS for Windows Release 6.0 SPSS Inc, Chicago IL

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CORRECTIONS

In the paper, "Understanding Cardiorespiratory Interactions: Cardiovascular Anatomy and Physiology" by Charles A Trant, Frank H Kern, and Jon N Meliones [Respir Care 1996;41(2):113-122], the authors state on page 114 that a normal ejection fraction is 75-80%. This range of values is only for infants and children. The normal ejection fraction for adults is approximately 55%.

The caption of Figure 2, page 125, in "The Role of Cardiorespiratory Interactions in Respiratory Care" by Donald R Black, Barbara G Wilson, and Jon N Meliones [Respir Care 1996;41(2):125-132] contains errors. The corrected caption is printed below with the corrections in italics.

The authors and editors are grateful to Marcel Lapointe, Chicoutimi Que, Canada, for raising the questions. We regret the errors.

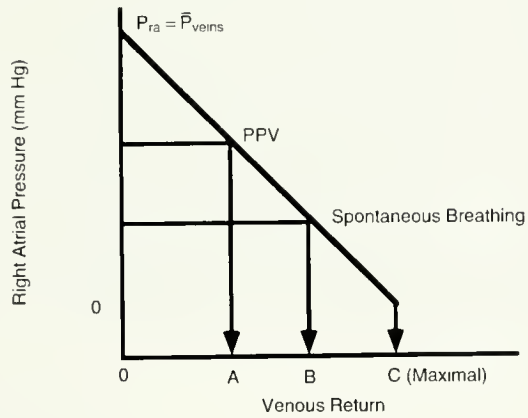

Fig. 2. Venous return to the right heart occurs passively and depends on a pressure gradient from the systemic veins to the right atrium. When right atrial pressure (P_{ra}) is zero, there is no impedance to flow back to the right heart, and venous return is maximal (*Point C*). As right atrial pressure *increases*, and mean systemic venous pressure ($\bar{P}_{veins}$) is held constant, there is a progressive reduction in venous return. When right atrial pressure exceeds mean systemic venous pressure, venous return ceases. During spontaneous breathing, right atrial pressure is low and systemic venous return is high (Point B). During positive pressure ventilation (PPV), intrathoracic pressure and P_{ra} increase resulting in reduced venous return (*Point A*). (Modified from Reference 2, with permission).

In the footnote of the Historical Notes: Effective Inhalation Therapy. Generalized Obstruction [reprinted in RESPIRATORY CARE 1996;41(3):233], Dr Edwin R Levine was noted to be an anesthesiologist. He was, in fact, a chest physician. We regret the error and thank Dr LeRoy Misuraca for bringing it to our attention.

Effectiveness of Adding Alternate-Day Theophylline to the Treatment Regimen of Patients with Moderate-to-Severe Asthma*

Ali Emad MD

BACKGROUND: The use of theophylline in the treatment of patients with asthma is controversial. MATERIALS & METHOD: We assigned 120 patients with moderate-to-severe asthma into 3 groups of equal numbers in order of their presentation. All study patients received inhaled beclomethasone (500 μg daily) and a short-acting inhaled β-2 agonist (salbutamol by metered dose inhaler) as necessary up to 3-4 times a day. In addition, patients in Group 2 (control group) were prescribed a placebo tablet, and patients in Group 3 received theophylline (Theo-Dur) 250 mg orally, every-other-day at bed time (10 PM). Groups 2 and 3 were blinded to their oral medication. The duration of the study was 6 months. The physician administering the pulmonary function tests was blinded to the study assignment of the patients. RESULTS: The means and standard deviations for the 3 groups for FEV_1, FVC, and PEFR on admission to the study did not vary significantly by ANOVA (FEV_1, $p = 0.718$; FVC, $p = 0.402$; PEFR, $p = 0.453$); whereas, at the close of the study the improvement in all 3 values for Group 3 was significant at $p = 0.0001$. The number of hospital admissions from Group 3 was also significantly lower than those of the other groups (ANOVA, $p = 0.0017$). CONCLUSION: Although the serum theophylline concentrations were probably below the therapeutic range because of the low, alternate-day dose (they were not measured), this study suggests that treatment of patients with moderate-to-severe asthma with theophylline may be beneficial. [Respir Care 1996;41(6):520-523]

Introduction

Asthma is a chronic inflammatory disease of the airways that can be severe enough to cause death.[1-3] Management of asthma is aimed mainly at lessening airway inflammation with anti-inflammatory drugs and reversing acute airway obstruction through the use of bronchodilators. The most effective anti-inflammatory drugs for the treatment of moderate-to-severe asthma are corticosteroids;[4-8] and short-acting inhaled β-2 agonists are used as necessary in the chronic management of asthma. The use of theophylline is controversial.[9-13] Because theophylline has a narrow therapeutic range and many side effects, some authors recommend its use only after inhaled corticosteroids and inhaled β-2 agonists have failed to control the patient's symptoms.

The purpose of this study was to determine whether the regular, intermittent use of theophylline is beneficial in the management of patients with moderate-to-severe asthma and whether it is associated with clinically important side effects.

Methods & Materials

Patients

Data were collected from all patients referred to our clinic from a combination of complete physical examination, structured interview, and questionnaire administered by a physi-

The author is associated with the Department of Internal Medicine, Nemazee Hospital and Shiraz Medical Science University, Shiraz, Iran.

Correspondence: Ali Emad MD, Department of Internal Medicine, Nemazee Hospital, PO Box 71345-1414, Shiraz University of Medical Science, Shiraz, Iran.

*See the related editorial on pages 507-508.

cian specialist. The eligibility criteria included (1) age 18 years or older, with recurrent episodes of wheezing or dyspnea, (2) objective evidence of airways obstruction during episodes; (3) sufficient history available to categorize asthma severity; (4) objective evidence of improved airflow when symptom free.

Both male and female outpatients, aged 18 to 60, who had moderate-to-severe asthma[14] as defined by the American Thoracic Society[15,16] were evaluated; 120 subjects met our criteria and entered this 6-month study. Patients were excluded from the study if they had a history of cigarette smoking or use of oral corticosteroids. Those with occupational lung diseases (including occupational asthma), pulmonary fibrosis, and/or any systemic illness, and pregnant women and nursing mothers were also excluded. Although the prevalence of atopy was 15% in Group 1, 20% in Group 2, and 12.5% in Group 3, its presence was not a prerequisite for selection. Each individual gave informed and signed consent before the study. All patients were instructed in the correct way of using the metered dose inhaler (MDI) before the study.

Patients were assigned in order of presentation to one of three groups of equal size (ie, Patients 1, 4, 7, 10, 13, 16... to Group 1, Patient 2, 5, 8, 11, 14, 17... to Group 2, and so on.). All subjects received inhaled beclomethasone (500 μg daily) and short-acting inhaled β-2 agonist (salbutamol, by metered dose inhaler) as necessary up to 3-4 times a day. Patients in the control group (Group 2) also received a placebo tablet and patients in Group 3 received sustained-release theophylline tablets (Theo-Dur, Key Pharmaceuticals Inc, Kenilworth NJ) 250 mg orally at bedtime (10 PM) on alternate days. Subjects were blinded to the composition of the tablets.

Pulmonary Function Tests

Pulmonary function testing was performed for all patients at the beginning of the study, during their monthly clinic visits, and at the termination of the clinical follow up 6 months later. All tests were done by a single physician (blinded to the treatment assignment), using a FUDAC-50 spirometer (Fukunda Sangyo Co Ltd, Japan). Forced expiratory volumes (FEV_1)and flows were determined at least 3 times at 1-minute intervals, with the patients seated upright. The effort yielding the best FEV_1 and peak expiratory flow (PEFR) was chosen as maximal.

Follow-Up & Evaluation

Duration of the study was 6 months. All subjects had regular monthly follow-ups and spirometric evaluations. The numbers of hospital admissions due to severe attacks of asthma 6 months prior and 6 months after the study were also recorded. Each attack was managed with inhaled short-acting β-2 agonist (salbutamol metered dose inhaler) 2 to 3 puffs every 3-4 hours for the first 24-48 hours. The patients, however, were instructed to be admitted to a predetermined hospital for management of their attacks, if they did not respond to inhalation of β-2 agonist at any time during this period.

During their hospital admissions, they were managed with oxygen, inhaled β-2 agonist, hydration, and, if necessary, a short-course of intravenous hydrocortisone, which was tapered rapidly over a 3-day period and then discontinued. No patient needed oral corticosteroids. No patients were admitted for more than 3 days.

Data Analysis

Statistical analysis was done using ANOVA. A Newman-Keuls test was used to examine response differences among the means of the three groups for ANOVA, with p values significant at $p < 0.05$.

Results

The 3 groups of subjects with moderate-to-severe asthma were evaluated. The characteristic features of all groups are shown in Table 1. The 3 groups were comparable in regard to FEV_1, FVC, PEFR at the beginning of the study (Table 2). The values at the end of the study (6 months) are shown in Table 3. The mean (SD) PEFRs at the beginning of the study are shown in Table 2. The values of the same variables measured 3 and 6 months after the beginning of the study are shown in Tables 3 and 4. Patients in Group 3 (the treatment group) showed a significant improvement at 3 months but a much higher improvement at 6 months compared to the other 2 groups.

Table 1. Characteristics of Subjects Meeting Study Criteria

	Group 1 (n = 40)	Group 2 (n = 40)	Group 3 (n = 40)
Age (years)	32.32 (6.95)*	34.47 (5.26)	34.52 (5.52)
Men	12	17	11
Women	28	23	29

*Mean ± SD

The numbers of hospital admissions for each group 6 months prior to the study, during the study, and during the 6 months following the study are shown in Table 5. None of the patients in Group 3 suffered from any major side effect or any toxicity from the theophylline. The only reported side effect of the theophylline was a minimal tremor in 2 patients.

Discussion

After more than 50 years of clinical use, the mode of action of theophylline is still poorly understood. It is a methylxan-

Table 2. Results of Pulmonary Function Testing in All Groups Prior to the Study*

	FEV_1 (L)	FVC (L)	PEFR (L/s)
Group 1 (n = 40)	1.35 (0.13)	2.26 (0.13)	3.02 (0.44)
Group 2 (n = 40)	1.32 (0.19)	2.23 (0.13)	2.91 (0.51)
Group 3 (n = 40)	1.31 (0.19)	2.22 (0.13)	2.87 (0.51)
ANOVA	p = 0.718	p = 0.402	p = 0.453

*All groups received medication by inhalation; Group 2 also received alternate-day placebo tablet; Group 3 received alternate-day theophylline. FEV_1 = volume exhaled in first second of forced expiration; FVC = forced vital capacity; PEFR = peak expiratory flow. Values are mean (SD).

Table 3. Results of Pulmonary Function Testing in All Groups 3 Months after Beginning of the Study*

	FEV_1 (L)	FVC (L)	PEFR(/s)
Group 1 (n = 40)	1.40 (0.19)	2.30 (0.06)	3.08 (0.19)
Group 2 (n = 40)	1.41 (0.19)	2.31 (0.13)	3.09 (0.13)
Group 3 (n = 40)	1.48 (0.06)	2.40 (0.19)	3.12 (0.19)
ANOVA	p = 0.03	p = 0.02	p = 0.04

*All groups received medication by inhalation; Group 2 also received alternate-day placebo tablet, Group 3 received alternate-day theophylline. FEV_1 = volume exhaled in first second of forced expiration; FVC = forced vital capacity; PEFR = peak expiratory flow. Values are mean (SD).

Table 4. Results of Pulmonary Function Testing in All Groups at the End of Study (6 Months)*

	FEV_1 (L)	FVC (L)	PEFR (L/s)
Group 1 (n = 40)	1.51 (0.19)	2.38 (0.19)	3.29 (0.38)
Group 2 (n = 40)	1.49 (0.19)	2.35 (0.19)	3.12 (0.38)
Group 3 (n = 40)	1.82 (0.13)	2.80 (0.19)	4.06 (0.57)
ANOVA	p = 0.001	p = 0.001	p = 0.001

*All groups received medication by inhalation; Group 2 also received alternate-day placebo tablet, Group 3 received alternate-day theophylline. FEV_1 = volume exhaled in first second of forced expiration; FVC = forced vital capacity; PEFR = peak expiratory flow. Values are mean (SD).

Table 5. Hospital Admissions for All Groups for 3 Time Periods

	Number of Admissions		
	6 Months Prior to Study	During 6 Months of Study	6 Months following Study
Group 1 (n = 40)	17	15	14
Group 2 (n = 40)	20	13	18
Group 3 (n = 40)	12*	9†	4‡

All groups received medication by inhalation; Group 2 also received alternate-day placebo tablet; Group 3 received alternate-day theophylline. *p = 0.188; †p = 0.340; ‡p = 0.0017.

thine similar in structure to the dietary xanthines caffeine and theobromide.

Theophylline has a narrow therapeutic index, potential toxicity, and frequent side effects even at serum concentrations considered to be therapeutic.[17] Thus, it has been suggested that it be used only if treatment with ideal doses of inhaled steroids and bronchodilators fails to provide adequate control of symptoms.[18,19] To avoid the side effects of theophylline, at least one group of investigators has suggested a conservative therapeutic range of 5-15 μg/mL as the ideal serum theophylline concentration (STC).[20]

Because we used a low dose, the STC in our patients should have been lower than this therapeutic range; however, the last group who received theophylline (intermittently but regularly) had more improvement in their FEV_1 (ANOVA, p = 0.0001) and PEFR (ANOVA, p = 0.0001) and required fewer hospital admissions (ANOVA, p = 0.0017) compared to the other groups at termination of the study. The differences in changes in FEV_1, PEFR, and hospital admissions were also significant.

This study suggests that alternate-day administration of theophylline may be beneficial, producing improved FEV_1 and PEFR with no major or significant side effects and significantly reducing the number of hospitalizations of these patients as well. The beneficial effects of intermittent regular treatment with theophylline may be multifactorial. Theophylline has preventive effects on nocturnal attacks of asthma[21-23] and may decrease the bronchial hypersensitivity.

It has been well shown that theophylline can reduce the inflammatory process of asthma even at levels below its therapeutic range.[24,25] In other words, it is able to reduce asthma symptoms at a lower serum level than has been advocated for bronchodilatation. It may act directly on the permeability-regulating microvascular endothelial cells and inhibit both neural and nonneural exudative responses into tracheobronchial airways.[26]

There is also good evidence that theophylline and β-agonists have additive effects, even if true synergy is not seen.[27,28]

It is, therefore, possible that if adequate bronchodilation is not achieved by β-agonists alone, theophylline, even at low oral doses could be beneficial by inducing further bronchodilation.[29]

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What Is Tracheal Pressure-Triggering?—and Do We Need It?

YES! We Do Need It!*

With assisted or supported breaths, patients and ventilators interact during each phase of the ventilatory cycle: the triggering phase, the gas-delivery phase, and the breath-termination, or cycling phase. Suboptimal interactions during any of these phases can lead to imposed muscle loading. These imposed loads may lead to muscle fatigue, dyspnea, increased need for sedation, and even ventilator dependence.

A number of recent enhancements have improved patient-ventilator interactions. Among these are more sensitive and responsive demand valves (including the use of flow-triggering); pressure-targeted breaths delivering patient-controlled flows; the capability to adjust the rate of rise (or rise-time) of these pressure-limited breaths to better match patient demand at the initiation of gas flow; and capabilities to adjust the duration of inspiration.

A common feature of all interactive breaths is that the ventilator must have a means of detecting patient effort. On many ventilators, a pressure or flow sensor located somewhere in the ventilator circuitry detects the changes in pressure or flow to which the ventilator responds with changes in gas flow. Banner et al[1] in a 1993 paper introduced the idea of placing a pressure sensor at the tip of the endotracheal tube (tracheal sensor) rather than in the ventilator circuitry. They argued that placing the ventilator sensor closer to the site of patient effort would reduce breath-triggering work, thereby improving patient-ventilator interactions, and they presented data from a lung model supporting this contention. In this issue of RESPIRATORY CARE, Bhatt et al[2] also use a lung model to study the effects of a tracheal pressure sensor, but they conclude that the reduction in triggering work can be mimicked by simply applying airway pressure support. However, I believe that this conclusion may be too simplistic and that tracheal pressure sensing deserves more study.

To understand why tracheal pressure sensing might improve patient-ventilator triggering interactions differently than does simply adding airway pressure support, one needs to consider the properties of demand valves and endotracheal tubes. Demand valves are characterized by both sensitivity (the amount of pressure or flow change that must be sensed before the valve is opened), and responsiveness (the speed at which the valve opens to reach the target flow or pressure). Valve function is also affected by the endotracheal tube, which serves as a flow-resistive element separating the lungs (where patient effort is generated) from the traditional ventilator sensing sites in the circuitry.

Moving the pressure sensor to the patient side of the endotracheal tube has little effect on the sensitivity of the valve because, with a pressure-triggering system, the demand valve is closed when patient effort to initiate a ventilator breath begins. Under these circumstances, pressure changes move roughly at the speed of sound and thus moving the sensing site a few centimeters closer to the pleura and muscles has minimal effect. (The sensitivity of a flow-based trigger, of course, is not affected by pressure-sensor location).

However, moving the pressure sensor to the patient side of the endotracheal tube could have substantial effect on responsiveness of the valve, especially with a pressure-targeted breath. This is because the pressure-targeted breath is designed to have the demand valve provide enough flow to achieve a so-called square wave of inspiratory pressure at the sensing site. If the sensing site is in the ventilator circuitry, the resistive nature of the endotracheal tube converts this square-wave airway-pressure profile into a so-called ramped pressure profile in the trachea (Fig. 1, left panel). This pattern may not provide a rise in alveolar pressure rapid enough to satisfy active patient demand. Moreover, increasing the magnitude of the airway inspiratory pressure does not change the ramp pattern in the lung. On the other hand, placing the demand-valve pressure target beyond the endotracheal tube results in the development of the square pressure profile in the trachea and a decelerating pressure profile in the ventilator circuit (Fig. 1, right panel). Thus, contrary to the conclusion of Bhatt et al,[2] tracheal targeting may not be simply inadvertent airway pressure support.

*For an opposing viewpoint, see page 526.

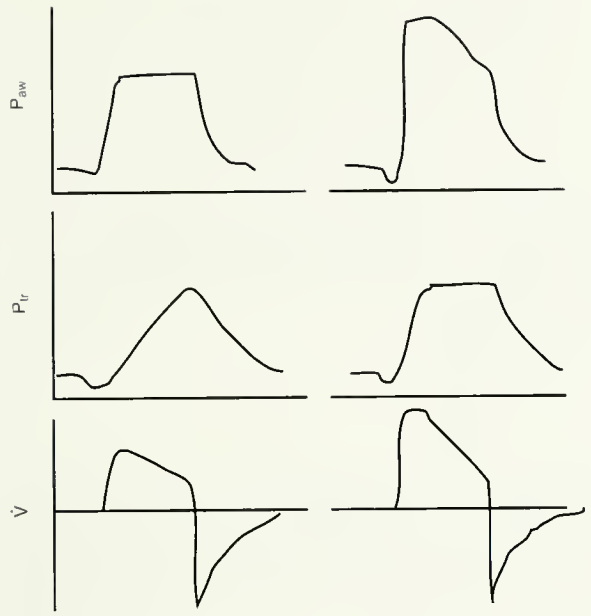

Fig. 1. Airway pressure (P_{aw}), tracheal pressures (P_{tr}), and flow ($\dot{V}$) patterns when pressure support is targeted to the airway (left panel) or targeted to the trachea (right panel). When the pressure target is the airway, the resistance of the endotracheal tube produces a ramped pressure wave form in the trachea. In contrast, when the pressure target is the trachea, the ventilator must respond with a higher flow and a higher pressure in the circuit to overcome endotracheal tube resistance and produce the desired square wave in the trachea.

The measurements made by Bhatt et al may not allow these points to be illustrated. Specifically, work was calculated only at the pressure-sensing site. Thus, work using tracheal sensing was measured in the trachea, but work during airway sensing was measured in the circuit. That they were similar at the ventilator's pressure-sensing site under both conditions is not surprising. I believe that measurements at both sites under both conditions would have been more appropriate to illustrate the concepts outlined and illustrated in Figure 1.

In conclusion, the more rapid rise of pressure in the trachea from tracheal sensing could conceivably enhance patient-ventilator synchrony during the initial phases of breath initiation. It is clear that this needs to be studied clinically, and proper adjustments of the rate of rise of pressure in the trachea need to be titrated to patient demands. Distal pressure-sensing and breath-targeting may have distinct advantages in improving patient-ventilator synchrony. It is premature to dismiss it as merely "inadvertent" pressure support.

Neil R MacIntyre MD
Professor of Medicine
Respiratory Care Services
Duke University Medical Center
Durham, North Carolina

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What Is Tracheal Pressure-Triggering?—and Do We Need It?

NO! We Don't Need It!*

Optimizing the ability of the patient to trigger the ventilator is a common goal for clinicians and manufacturers alike. For clinicians, setting appropriate sensitivity, adjusting rise time, and eliminating unnecessary causes of the imposed work of breathing are orders of the day. For manufacturers, mechanical and software engineering to speed signal processing, improve sensor response, and maximize the post-trigger phase are important goals.

Issues related to triggering and the work of breathing have been the subject of frequent reports in RESPIRATORY CARE. Sassoon and Mahutte covered the major technical aspects of pressure and flow triggering in the March 1995 issue.[1] Their conclusions are in agreement with mine and others.[2-4] That is, flow-triggering does appear to have some advantage over pressure-triggering, related to a faster response time. The open position of the exhalation valve during flow-triggering may improve response compared to the position of the exhalation valve (closed) during pressure-triggering. However, the major difference in flow-triggering systems appears to be the post-trigger phase or, perhaps more appropriately, the limit and cycle variables. In fact, most current-generation ventilators provide 1.5-3.5 cm H_2O pressure support during spontaneous breathing with continuous positive airway pressure (CPAP).[5,6]

Studies comparing current-generation mechanical ventilators demonstrate a minute amount of imposed work of breathing during spontaneous breathing.[5,6] In many cases these values are reported in millijoules and are approximately 2% of the normal work of breathing (0.5 mJ/L). A number of investigators agree that the difference in the imposed work of breathing between ventilators is measurable, but of little clinical importance.[5,6]

The question raised then becomes, What next? Over the past several years, Banner and colleagues[7-10] have suggested triggering the ventilator from the carinal tip of the endotracheal tube. This technique eliminates the imposed work of breathing caused by the endotracheal tube.[11] In concept, this appears to make sense. Moving the trigger point close to the source of the signal should improve the response. In fact, the work imposed by the endotracheal tube is 10 times that imposed by the ventilator's trigger mechanism. The ability to overcome endotracheal tube resistance might prove useful in weaning patients vulnerable to respiratory muscle fatigue.

Banner et al[8] reported that tracheal pressure-triggering reduces the imposed work by 382% and 315%, compared to proximal pressure- and flow-triggering.[8] A series of articles by this group of investigators has continued to show the superiority of tracheal pressure-triggering.[7-10] But, is it tracheal pressure-triggering that is causing the reduced work of breathing?

Each breath delivered from the ventilator can be described by the trigger, limit, and cycle variables. Tracheal pressure-triggering also includes tracheal pressure-limiting and tracheal pressure-cycling. The end result is demonstrated nicely in a figure (Fig. 1) from Banner et al's second paper.[8] The figure demonstrates that when using the tracheal pressure signal to control breath delivery, positive airway pressure is present at the proximal airway. The 'magic' behind tracheal pressure-triggering then is the use of pressure support ventilation! Tracheal pressure-triggering may reduce response time slightly, but how much faster than 80 ms does the response time need to be? Now, before my pediatric colleagues jump in, I am referring to adult patients with respiratory frequencies in the range of 12-30 breaths/min. In neonates breathing 80 times a minute, even a slight improvement in response time may be helpful.

In this issue of RESPIRATORY CARE, Bhatt et al[12] present their work related to the imposed work of breathing during CPAP and the role of tracheal pressure-triggering. Their work quite nicely supports my contention that tracheal pressure-triggering is a complex way of providing pressure support to overcome endotracheal tube resistance. I quote from their paper, "The decrease in imposed work during tracheal pressure-triggering is not due to an alteration in the triggering func-

*For an opposing viewpoint, see page 524.

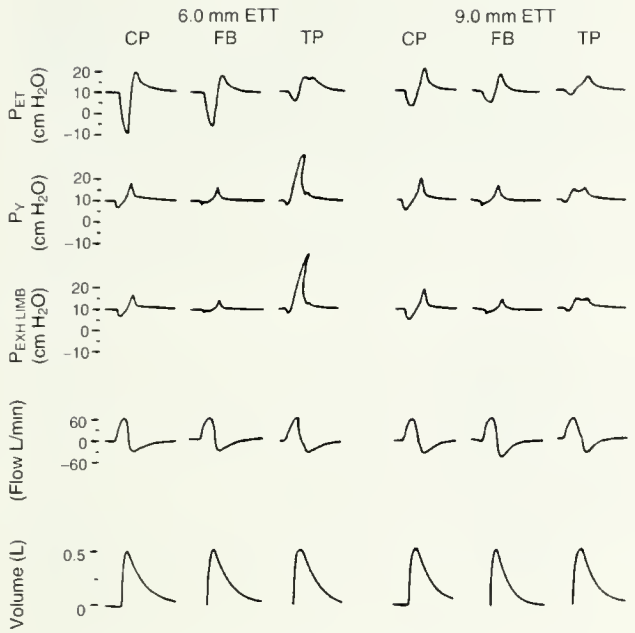

Fig. 1. Pressure changes measured at the tracheal end of the endotracheal tube (P_{ET}), Y piece (P_Y), and exhalation limb ($P_{EXH\ LIMB}$) of the breathing circuit, and changes in inspiratory and expiratory flows and volume during spontaneous breathing with conventional pressure-triggering (CP), flow-by triggering (FB), and tracheal pressure-triggering (TP) with 6- and 9-mm internal diameter endotracheal tubes (ETT). Note that greater decreases in inspiratory pressure were measured at the tracheal end of the endotracheal tube than at the Y piece or exhalation limb of the breathing circuit, and that pressure decreases were greatest during conventional pressure-triggering, less during flow-by triggering, and lowest during tracheal pressure-triggering under the same conditions of peak inspiratory flow demand and tidal volume. Under these conditions, inspiratory imposed work by the breathing apparatus was significantly ($p < 0.05$) lower during flow-by triggering compared with conventional pressure-triggering, and during tracheal pressure-triggering compared with flow-by triggering and conventional pressure-triggering.

tion per se, but rather due to an inadvertent pressure assist, and is no more advantageous than the application of pressure support to compensate for the apparatus work." Well said. I am always happy to praise the work of others—particularly when it supports my opinion.

Where does this leave us? In an opposing opinion, MacIntyre[13] tells us that tracheal pressure-triggering (limit and cycling) is the next logical step in reducing imposed work. I do not agree. As Bhatt et al have shown, tracheal pressure-triggering is not only a misnomer but also is no more effective than pressure support. I loathe adding another term to the morass of mechanical ventilation terminology, but tracheal pressure control is a more accurate term.

Several authors have attempted to develop equations to predict the amount of pressure support necessary to overcome endotracheal tube resistance, given a known tube size and patient flow demand.[14,15] This is the simple man's tracheal pressure control. One might wonder why I think tracheal pressure control is unnecessary. Consider the cost and complexity of adding tracheal pressure control to a current-generation ventilator. An additional pressure sensor and software to support that sensor will be required. Protection of the signal from humidity and secretions will be necessary. Will we need special endotracheal tubes with imbedded lumens for the new sensor? Will a separate catheter be advanced to the distal tip of the endotracheal tube, further increasing the imposed work by reducing internal diameter and increasing turbulence? How will position of the catheter be determined and maintained? How will the catheter affect the ease and effectiveness of endotracheal suctioning? If the tracheal pressure signal fails, what will happen? Redundant systems for failure modes will be required. In addition, has anyone shown that overcoming endotracheal tube resistance is either helpful or a good idea? Certainly, in cases of inappropriately sized airways the tracheal tube imposes an inordinate amount of work.[16] But what about the probable millions of patients who have been successfully weaned using T-piece and CPAP trials, breathing through the endotracheal tube without pressure support? Any number of maneuvers and manipulations can reduce the work of breathing. We just do not know which ones are important or useful. Under these circumstances, tracheal pressure control seems like a 'long haul for a short slide.'

As an alternative, Guttmann et al[17] have developed a technique that allows continuous calculation of intratracheal pressure and automatic compensation of imposed work. By characterizing the flow-resistive properties of endotracheal tubes of different sizes and measuring flow, these investigators were able to successfully mimic tracheal pressure control. However, the numerous disadvantages of tracheal pressure control and monitoring are relieved. In this situation, the clinician enters the tracheal tube size into the ventilator's logic and the ventilator automatically compensates for tube resistance. Of course this also requires modification of ventilator software, but it appears to me to be a simpler and more reliable method. This of course does not eliminate the questions surrounding the value of eliminating tracheal tube resistance, but that is the subject of further study. As an example, Figure 2 demonstrates pressure-volume curves from the proposed system. In this figure, the actual measured tracheal pressure is compared to the calculated pressure and proximal airway pressure. This demonstrates the utility of a 'smart' ventilator's performing calculations and eliminating imposed work without further clinician input.

I admit that automatic compensation for endotracheal tube resistance is in the future, but it is certainly in the realm of tasks that current-generation ventilators are capable of performing. Tracheal pressure control, not pressure-triggering, may have some advantage, but is fraught with technical prob-

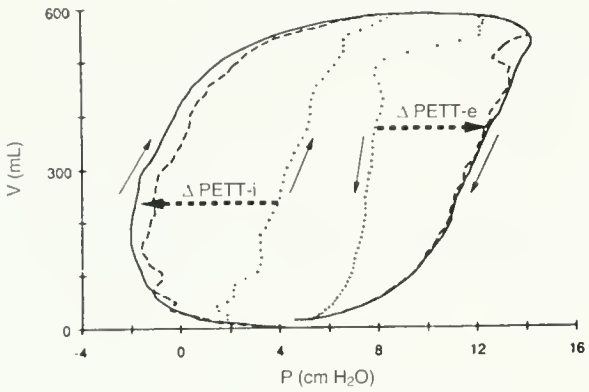

Fig. 2. Pressure-volume curves from ventilator system that continuously calculates intratracheal pressure and automatically compensates for imposed work. Actual measured tracheal pressure (——) is compared to calculated pressure (– – – –) and proximal airway pressure (·····).

lems. As for me, I'll just keep delivering pressure support the old-fashioned way.

Richard D Branson RRT
Assistant Professor of Surgery
University of Cincinnati
Cincinnati, Ohio

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Laboratory and Clinical Experience with Liquid Ventilation in Respiratory Failure

Ronald B Hirschl MD

Introduction
Partial vs Total Liquid Ventilation
Gas Exchange & Pulmonary Function
Effects on Lung Injury & Inflammation
Clinical Experience
Dosing & Distribution
In Summary

Introduction

The concept that one could breathe liquids yet sustain gas exchange was first demonstrated by Kylstra et al[1] in 1962, when they documented the ability of mice to survive while submerged in saline (0.9% NaCl) that has been oxygenated under hyperbaric conditions. Clark and Gollan[2] subsequently led the way toward the use of perfluorocarbon for liquid breathing when they reported the ability to sustain gas exchange in the submerged, spontaneously perfluorocarbon-breathing mouse. Perfluorocarbons are structurally similar to hydrocarbons with the hydrogen replaced by fluorine. The carbon chains vary in length, and an additional moiety often is attached to the molecule that, together, give unique properties to each perfluorocarbon. In general, perfluorocarbons have excellent oxygen and carbon dioxide carrying capacity (O_2, 50 mL/dL and CO_2, 160-210 mL/dL).[3] They are clear, odorless, inert liquids that are immiscible in aqueous and most other solutions. They are relatively dense (1.7-1.9 g/mL) and have a low surface tension (15-19 dyn/cm).

Partial vs Total Liquid Ventilation

The work of breathing is markedly increased during spontaneous perfluorocarbon breathing because of the elevated resistance to flow of a liquid in the airways. For this reason, mechanical devices have been developed and tested in the laboratory to provide total liquid ventilation (TLV) during which the lungs are first filled to a volume equivalent to the functional residual capacity (FRC, approximately 30 mL/kg) and then ventilated with perfluorocarbon. In 1974, Shaffer and Moskowitz[4] documented the ability of such a device to provide demand-regulated TLV. Other devices have been developed that apply extracorporeal technology (Fig. 1).[5]

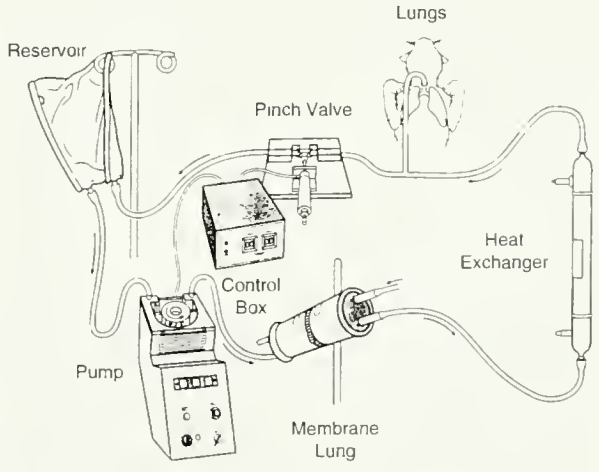

Fig. 1. A flow-limited, time-cycled device for liquid ventilation. The control box allows variation in inspiratory and expiratory time. During inspiration, the pinch valve is occluded, and the pump functions, so that a tidal volume is generated. During expiration, pump function is discontinued, occluding the inspiratory limb. At the same time, the pinch valve releases, allowing drainage of perfluorocarbon into the reservoir. (From Reference 5, with permission.)

Dr Hirschl is Assistant Professor, Departments of Surgery, CS Mott Children's Hospital and the University of Michigan, Ann Arbor, Michigan.

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Correspondence: Ronald B Hirschl MD, F3970 Mott Childrens Hospital, 1500 E Medical Center Drive, Ann Arbor MI 48109-0245.

In 1991, the first experience with partial liquid ventilation (PLV) in a healthy rabbit model was reported.[6] With this technique the lungs are filled with perfluorocarbon to a volume equivalent to FRC and then gas ventilated with a standard mechanical ventilator. The adequacy of the perfluorocarbon dose is assessed, during PLV, by observing a meniscus of perfluorocarbon within the endotracheal tube at end-expiration. PLV has advantages over TLV: PLV does not require use of an additional device or an understanding of the physics of liquid flow through the airways, endotracheal tube, and liquid-ventilation device. Therefore, PLV can be relatively easily performed if one has an understanding of the management of critically-ill patients with respiratory failure. A number of studies have demonstrated the efficacy of PLV in improving gas exchange in preterm and full-term neonatal, pediatric, and adult lung-injury models created by intravenous oleic-acid administration, gastric-acid aspiration, meconium aspiration, congenital diaphragmatic hernia (CDH), and saline lung lavage.[7-13]

Gas Exchange & Pulmonary Function

The mechanisms by which gas exchange and pulmonary function are increased during liquid ventilation have been explored over the last few years. We previously demonstrated the ability of TLV to recruit collapsed lung regions in an ex-vivo lung model of atelectasis.[14] Compared to gas ventilation, end-expiratory lung volume increased 8-fold in otherwise normal, atelectatic lungs and 14-fold in surfactant deficient, atelectatic lungs during TLV. These data suggest that perfluorocarbon might enhance alveolar recruitment in the setting of atelectasis with or without surfactant deficiency and, thereby, improve pulmonary gas exchange. We subsequently evaluated the ability of both TLV and PLV to resolve the atelectasis/consolidation that is commonly seen in the dependent regions of the lungs in patients with ARDS.[15] Perfluorocarbon appears to effectively distribute into those dependent lung regions during both PLV and TLV, with associated alveolar recruitment.[16,17] Gauger et al[18] evaluated FRC in an oleic-acid model of lung injury during PLV and gas ventilation. FRC was measured by helium dilution and by a technique similar in concept to body plethysmography. The gas FRC decreased following induction of lung injury and further decreased following initiation of PLV. However, when one added the gas FRC to the volume of perfluorocarbon administered, the mean total FRC in the PLV animals was equal to or greater than that observed before the injury. These data suggest that administration of perfluorocarbon during PLV results in the recruitment of otherwise atelectatic or consolidated lung regions and may enhance ventilation-perfusion matching and gas exchange. In addition, administration of perfluorocarbon tends to result in redistribution of pulmonary blood flow away from those dependent regions toward the nondependent, better-aerated lung regions during TLV.[19] This pattern of redistribution of pulmonary blood flow may also result in an improvement in ventilation-perfusion matching and gas exchange during PLV following lung injury.

Effects on Lung Injury & Inflammation

One fascinating and fairly reproducible finding is a reduction in the degree of lung injury and inflammation observed during liquid compared to gas ventilation in animal models of respiratory failure.[9,20] Studies have now documented a decrease in neutrophil infiltration, intra-alveolar hemorrhage, and exudate formation during PLV compared to gas ventilation of rats with lungs injured by cobra venom factor.[21,22] The basis for this reduction in lung injury is unclear. A lavage effect during TLV or PLV or a mechanical effect due to the presence of the highly immiscible perfluorocarbon in the reinflated alveoli are all possible mechanisms for this observation. In addition, there may be a direct anti-inflammatory effect provided by the presence of perfluorocarbon in the alveolus—in vitro studies have suggested that macrophage function and neutrophil adhesion and cell killing may be reduced in the presence of perfluorocarbon.[23-25] Further clinical investigation is required to ascertain the role that this protective effect may play in preventing initial development and progression of lung injury in patients with potential or established respiratory insufficiency.

Clinical Experience

Phase I and II studies evaluating the efficacy of PLV in premature newborn, pediatric, and adult patients with respiratory insufficiency have been completed and have demonstrated encouraging results. A multicenter study[26] involving 13 premature newborns who had failed two doses of surfactant and had a $P_{aO_2}/P_{AO_2} < 0.2$ demonstrated a 2-fold increase in compliance from approximately 0.2 to 0.4 mL · cm H_2O^{-1} · kg^{-1} and a decrease in oxygenation index (OI = [(mean airway pressure · F_{IO_2})/P_{aO_2}]100) from approximately 50 to 10 during the 24-hour period following initiation of PLV with perflubron (LiquiVent®, Alliance Pharmaceutical Corp, San Diego CA).

In similar phase I and II studies, we evaluated parameters of pulmonary function and gas exchange in adults, children, and newborns with respiratory failure on extracorporeal life support (ECLS).[27] This series of patients included 10 adults with a variety of diagnoses (pneumonia = 7, charcoal aspiration = 1, ARDS = 1, and asthma = 1); 4 pediatric patients (pneumonia = 2, hydrocarbon aspiration = 1, ARDS = 1); and 5 neonates (CDH = 4, primary pulmonary hypertension of the newborn = 1). Figure 2 is the anteroposterior chest radiograph of an adult with ARDS and Figure 3 that of a newborn with congenital diaphragmatic hernia, after the lungs have been filled with the radiopaque perflubron.[28] As demonstrated in Figures 4 and 5, a decrease in the difference between alveolar and arterial P_{O_2} and an increase in static pulmonary com-

Fig. 2. Representative anteroposterior radiograph of a 36-year-old adult with pneumonia. (From Reference 29, with permission.)

Fig. 3. Chest radiograph from a neonate with congenital diaphragmatic hernia during partial liquid ventilation. (From Reference 28, with permission.)

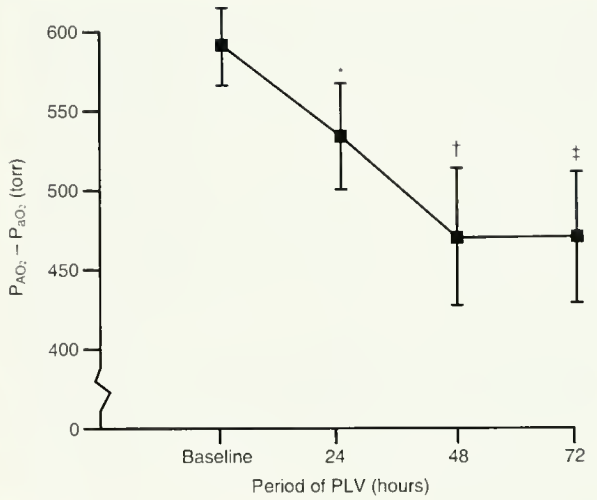

Fig. 4. Mean change in the alveolar-arterial P_{O_2} difference prior to and during partial liquid ventilation in 19 adults, children, and neonates with respiratory failure, during periods of discontinuation of extracorporeal life support. $p = 0.0002$ by repeated measures ANOVA; * $p = 0.006$, † $p = 0.003$, ‡ $p = 0.002$ by post-hoc paired *t* test with Bonferroni correction $p = 0.017$. (From Reference 27, with permission.)

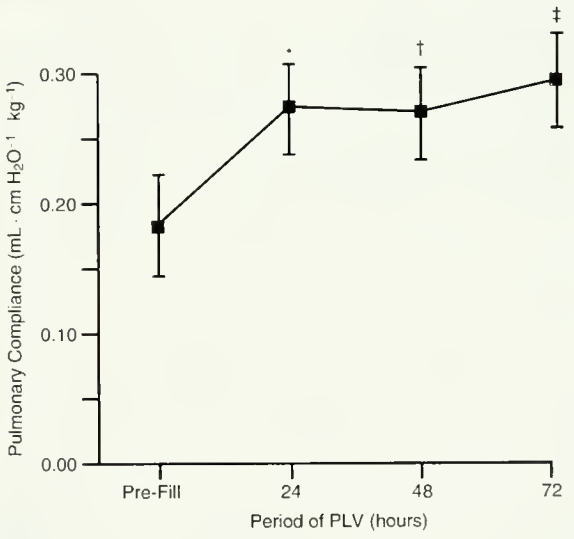

Fig. 5. Mean change in static pulmonary compliance, corrected for patient weight, prior to and during partial liquid ventilation in 19 adults, children, and neonates with respiratory failure during periods of discontinuation of extracorporeal life support. $p = 0.0002$ by repeated measures ANOVA; * $p = 0.002$, † $p = 0.003$, ‡ $p = 0.01$ by post-hoc paired *t* test with Bonferroni correction $p = 0.017$. (From Reference 27, with permission.)

pliance were observed during periods of discontinuation of ECLS in the 72 hours following initiation of PLV.[27] Similar improvements were observed in individual adult, pediatric, and neonatal populations.[28-30] All patients tolerated administration of perflubron without evident hemodynamic compromise. Of the 19 patients, 14 were successfully weaned from extracorporeal support, and 11 survived to discharge. Pneumothoraces occurred during PLV in 9 patients, although 6 of these had ipsilateral pneumothoraces prior to initiation of PLV.

Perfluorocarbon was observed in the chest cavity in 3 patients with pneumothoraces and was noted on subsequent radiographs to resolve slowly without evident deleterious effects. One neonate developed pulmonary hemorrhage approximately 2 days after discontinuation of ECLS and 4 days after administration of the last dose of perflubron. Mucus plug formation that compromised gas exchange was observed in one patient and resolved with aggressive suctioning and bronchoscopy. Although a number of other complications of critical disease processes and ECLS were observed in these critically ill patients, none appeared to be related to PLV. Repeated bronchoscopy in all patients failed to demonstrate any evidence of adverse effects upon the visible airways. The 11 surviving patients were well, without evidence of pulmonary or systemic adverse effects from the use of perfluorocarbon after, 8 to 18 months of follow up.

Dosing & Distribution

Protocols that are presently active involve adults, children, full-term newborns (non-CDH), CDH newborns, and preterm newborns who are not on ECLS. In these studies, perfluorocarbon is administered as frequently as necessary, but in general every 2-4 hours, to maintain liquid volume equivalent to FRC. The fill volume is confirmed by observing a meniscus within the endotracheal tube at end-expiration during transient ventilator disconnection. In some patients, however, gas exchange, tidal volume, and hemodynamics are optimal at a dose less than that necessary to fill to FRC. It is important to monitor tidal volume during dosing—a significant decrease in tidal volume indicates that dosing should be stopped until peripheral distribution of the perfluorocarbon with alveolar recruitment causes an improvement in compliance. Examination of pressure-volume curves during liquid ventilation reveals a hydrostatic effect during early inspiration, which we have referred to as 'the beak.'[31] This hydrostatic effect is only observed when perfluorocarbon fills the central airways at end-expiration and, therefore, indicates that the lungs are filled to, or near to, FRC. Figure 6 shows the beak after dosing with 10 mL/kg perfluorocarbon in an adult patient. The beak disappears during the ensuing 60 minutes as the majority of the perfluorocarbon evaporates and/or distributes into regions of the lungs that were previously atelectatic or consolidated.[32] During initial and subsequent dosing, the presence of the beak can be used to indicate that the lungs are filled with perfluorocarbon to a volume approaching FRC.

In Summary

The technique of liquid ventilation is at an early stage in its evolution. There is much to be learned regarding optimal dosing, positioning, ventilator settings, and overall technique. However, the ability to effectively recruit alveoli and to improve pulmonary function and gas exchange through the use of perfluorocarbon presents an exciting intervention with the promise of enhancing our ability to effectively manage patients with respiratory failure.

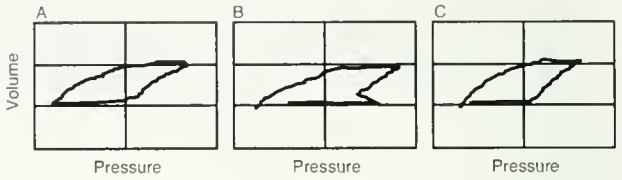

Fig. 6. Demonstration of the presence of the hydrostatic effect observed in the pressure-volume curve after filling with 10 mL/kg. This phenomenon has been termed the 'beak' and appears to be related to the presence of perfluorocarbon in the central airways at end-expiration. The presence of a beak may be used to monitor the degree of perfluorocarbon filling during initial and subsequent dosing. In this example, the beak is observed to resolve during the ensuing 60 minutes as the majority of the perfluorocarbon evaporates and/or distributes into regions of the lungs that had likely been atelectatic or consolidated. (From Reference 32, with permission.)

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Respiratory Care at the Crossroads—Evolution or Extinction?

Dean Hess PhD RRT

Introduction
Why Does U.S. Health Care Cost So Much?
Re-Engineering, Downsizing, & the Search for Excellence
Do We Need Respiratory Care Departments?
Keys to Survival
Hope for the Future

Introduction

I am humbled and flattered to have been invited to contribute to the Program Committee Lecture Series. In the past, these papers have provided important insights into the continued growth and development of the respiratory care profession. Respiratory care has been good for me both professionally and personally for nearly 25 years. I have made scores of friendships and developed many professional relationships. I cannot imagine anything I would rather do than practice respiratory care. With a personal investment of nearly 25 years and with 20 more years before retirement, I definitely want the respiratory care profession not only to survive, but also to prosper. As others have done with previous Program Committee Lectures,[1,2] I develop my suggestions for things that we should all be doing to ensure the survival of the profession.

As a profession, respiratory care has had an impressive growth over the past 40 years. From its humble beginnings—literally in the basement of many hospitals—respiratory therapists have evolved from on-the-job-trained technicians to formally educated and licensed bedside practitioners. Today's respiratory therapist is not only a technologist but also a skilled clinician and a physiologist as well. Under the umbrella of medical-staff-approved protocols, respiratory therapists in many hospitals implement and monitor respiratory care plans for acutely ill patients. Unlike 20 years ago, most textbooks used in respiratory care programs today are written by respiratory therapists. Respiratory therapists have also become involved in establishment of the scientific basis for respiratory care and publication in the peer-reviewed literature.

Health-care dollars were relatively plentiful during the formative years of the profession, allowing expansion of the roles and responsibilities of therapists with little concern for the cost of the care provided by these skilled individuals. In most hospitals in the United States, unlike most other countries, respiratory care is administered primarily by respiratory therapists. Health-care dollars are no longer plentiful. Prospective payment, managed care, capitation, and other programs are used increasingly to limit health-care costs. Consideration of charges, which once generated revenue, has been replaced by concern for costs. In fact, there is little relationship between costs and charges.[3] When revenue is fixed by prospective payment programs, increased costs can no longer be passed along to the consumer as increased charges.

Why Does U.S. Health Care Cost So Much?

It has almost become trite to say that health care in America is expensive. The rate of growth of health-care expenditures since 1970 has been 11.6%/year, whereas the rate of growth of the gross national product (GNP) has been 8.8%/year.[4] Health-care providers have been spending about 7% after inflation for new technology, increased illness severity, and quality improvements.[4] Health-care accounts for about

Dean Hess is Assistant Professor of Anaesthesia, Harvard Medical School, and Assistant Director of Respiratory Care, Massachusetts General Hospital—Boston, Massachusetts.

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Correspondence: Dean Hess, Respiratory Care, Ellison 401, Massachusetts General Hospital, Boston MA 02114.

15% of the GNP. In other words, the nation spends about one dollar of every six to provide health care.[5-7]

Most health care in the United States is financed by third-party payers, with little out-of-pocket expense to the patient. Thus, patients have little financial incentive to refuse any medical procedure. As a result, the cost of providing care has been driven by supply and not by demand. Until recently, third-party payers provided payment for all care that was deemed necessary and appropriate by the health-care provider. In other words, the more care that was provided, the greater the payment received. It has been estimated that the rate of growth in the cost of health care would be lower if patients had paid a greater percentage of the cost of that care themselves (Table 1).[7,8]

Table 1. Health Care's Share of the Gross National Product (GNP) for Different Levels of Third-Party Payment (Data are from Reference 7)

Third-Party (%)	GNP (%)
0	6.8
25	8.7
50	10.6
75	12.6
100	14.6

Much of the cost of health care for the elderly is provided by Medicare. As has been increasingly recognized over the past 20 years, Medicare in its current form is unsustainable for three reasons—demographics, technology, and third-party payment.[5] In the next 30 years, the proportion of Americans over the age of 65 will increase from 13% to more than 20% of the population, and the number of persons over the age of 70 will double. The fastest growing segment of the population are those over 85 years of age.[5,8] The older patients become, the more expensive is the health care that they consume. The same technologies that have extended life expectancy and the quality of life have also increased the cost of health care. It is not surprising that patients tend to consume more health-care services if someone else is bearing the cost.[8,9]

In spite of the high cost of health care in America, critics are asking increasingly whether we are getting our money's worth. About 15% of the population—45 million people—are not covered by a health insurance plan.[6] The principal tenets of health reform proposals have concentrated on cost and access; in other words, health care in America is too expensive and is not equally accessible to all citizens. Further, the emphasis of health care in America has been on acute care rather than preventive medicine. As a society, we smoke too much, eat too much, drink too much, and exercise too little—behaviors that result in disorders, such as cardiopulmonary diseases, that are expensive to treat in acute care hospitals.

Re-Engineering, Downsizing, & the Search for Excellence

Third-party payers (insurance companies and the government) are demanding lower costs for providing health care. This movement began when the Federal Government introduced diagnosis-related groups (DRGs) in the early 1980s in an attempt to control Medicare costs. Today the insurance industry is attempting to control costs with managed care programs designed to focus care on primary care providers, to avoid hospital admissions, and to shorten the length of stay when hospitalization cannot be avoided.[10-15] There is currently intense emphasis on transforming health-care financing. Systems are emerging that improve quality, lower costs, and enhance patient satisfaction, and, therefore, greatly affect the role of all health professionals in delivering care.[6]

One of the most visible transformations occurring in health care is so-called downsizing. Hospitals are closing, merging, and shrinking. At the Massachusetts General Hospital, the average daily census is projected to decrease from about 825 in 1992 to 625 in 1996. By the Year 2000, it has been projected that as many as half of the nation's hospitals will close, with loss of perhaps 60% of hospital beds. Further, a surplus of more than 100,000 physicians and more than 200,000 nurses is projected.[6] These changes in health-care delivery are truly revolutionary.

Not only is health care downsizing, but it is being re-engineered.[16,17] Re-engineering is defined as "...the fundamental rethinking and radical redesign of business processes to achieve dramatic improvements in critical, contemporary measures of performance, such as cost, quality, service, and speed."[18] The delivery of health care in the United States is being radically redesigned. Survival requires an understanding of the principal driving factors of this fundamental and radical redesign of health-care delivery—cost, access, quality, and patient satisfaction. Unlike any other time in the history of our profession, the quality and cost of respiratory care is in question—respiratory care is at a crossroads!

Nearly 15 years ago, Tom Peters suggested "eight basic principles to stay on top of the heap" in his popular book, *In Search Of Excellence*.[19] I believe it is important for us to consider these as tools for professional survival.

- *A bias for action.* Get involved and stay involved. Be part of the solution and not the problem. Do not sit back and let others decide your future. Do not hide from difficult decisions.
- *Staying close to the patient.* Never loose sight of the fact that our most important responsibility is to provide quality patient care. Be an advocate for the highest quality of patient care.
- *Autonomy and entrepreneurship.* Think independently. Have a passion for the respiratory care profession. Show ownership for the patient care that you provide.

- *Productivity through people.* Develop an essential interdependence with other health-care providers at the bedside.
- *Hands-on value-driven.* We must all (managers included) stay close to our values as a profession—quality, science-based patient care provided at the lowest possible cost.
- *Stick to the knitting.* Although we must become multi-skilled, we must also remember what has made us successful as respiratory therapists—a blend of technologist, clinician, and physiologist. As we broaden our scope, we must be careful not to move too far from our roots in respiratory care.
- *Simple form, lean staff.* As respiratory care professionals, we must add value without redundancy at the bedside. We must eliminate 'make-work' tasks that have questionable value or lack a scientific basis. Our respiratory care departments should be lean, with a minimum of supervisory levels.
- *Simultaneous loose-tight properties.* We must show the right blend of tough and gentle. We should strive to be tough-minded, but gentle-hearted in our dealings with others at the bedside.

Do We Need Respiratory Care Departments?

Respiratory care departments are no longer revenue-generating and have become cost centers. As such, the financial impact of a specialized department of respiratory therapists has become increasingly scrutinized. A respiratory care department adds to the expense of middle managers—department directors, assistant directors, and supervisors. It is recognized that many respiratory care procedures have been performed in the past by clinicians other than respiratory therapists, and some are currently performed by others in many hospitals. In some hospitals, respiratory care is unfortunately not viewed as a profession. There is the image of the respiratory therapist of the 1960s and early 1970s who was trained on the job, had a task orientation, and contributed little at the bedside beyond performance of low-level tasks. In an effort to control costs, the contribution of respiratory therapists to health-care delivery has become increasingly examined. Unfortunately, respiratory therapists and respiratory care departments have been eliminated from some hospitals (Fig. 1).

Respiratory care is here to stay. Aerosol therapy, chest physiotherapy, pulmonary function testing, and mechanical ventilation will continue to be part of the care of patients with pulmonary disease. However, respiratory care as a profession is at a crossroads. Does respiratory care need to be provided by respiratory therapists? What is the future of respiratory therapists? Are we evolving as a profession or are we doomed to extinction? What can we do to assure our survival? How does respiratory therapy fit into this era of managed care, restructuring, and patient-focused care?

I believe that a respiratory care department is necessary

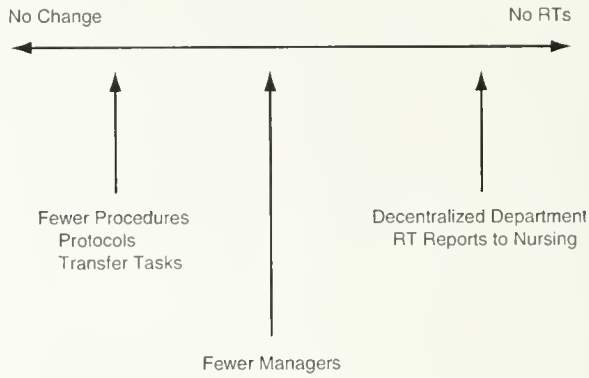

Fig. 1. The wide spectrum of effects of hospital restructuring on respiratory care.

to maintain the culture of respiratory care within the hospital. When the respiratory care department is decentralized, the level of respiratory care provided does not improve beyond its level at the time of decentralization. Continuing respiratory care education essentially stops, as does continuous quality improvement. When respiratory therapists no longer report to a central department, they stop talking with one another, and they no longer function as a learning organization.[20] Perhaps most important, decentralization of the respiratory care department is not efficient. For the same level of care, more resources (personnel and equipment) may be necessary for a decentralized department than a central department that can shift resources according to need. I strongly believe that decentralization of the respiratory care department is not good for resource management, the quality of respiratory care, or employee satisfaction.

Although I believe that a respiratory care department is important within a hospital, I do not believe that we need the numbers of managers and supervisors that are present in many departments. For a staff of skilled and mature professionals, managers and supervisors should not be needed in the traditional sense.[21,22] Managers are not needed to prepare the schedule, to prepare the payroll, or to conduct performance evaluations. Teams of therapists should be able to work together to prepare their own schedules and to conduct peer performance evaluations. Further, therapists can rotate the responsibility of team-leader or charge-therapist. The reason that respiratory care department managers are needed is not for people-management. Rather, the role of the manager is to provide leadership—professional, academic, clinical, and administrative. An important role of the respiratory care department manager is persuading upper-level hospital administrators that the contributions (financial and otherwise) of the department to the institution are valuable.[23] To do this effectively, managers must stay close to the customer—patients, therapists, physicians, nurses, and other health-care professionals.

Keys to Survival

I believe that we can do many things not only to survive, but also to cause the respiratory care profession to evolve. Allow me to share some of my opinions regarding issues that I believe are important for survival of the respiratory care profession (Table 2).

Table 2. Important Issues for the Survival of Respiratory Therapy.

- Add value at the bedside
- Move away from being solely task oriented
- Develop and maintain high-level skills
- Demonstrate flexibility
- Maintain a patient focus
- Provide professional leadership at the bedside
- Contribute to efforts to control health-care costs
- Develop an interdependence with other professionals at the bedside
- Expand the use of guidelines and protocols approved by the medical staff
- Upgrade the educational level of the profession
- Expand knowledge base through continuing education
- Become involved on hospital committees, in professional organizations, and in the community
- Work effectively as teams within the respiratory care department, the hospital, and the health-care delivery system
- Support the professional organizations that represent respiratory care

Adding Value

As respiratory therapists, we must always approach the bedside with something unique to contribute. We must demonstrate that quality of care suffers without our involvement. We are the only clinicians at the bedside with a strong background in technology and physiology (Fig. 2). Therefore, we must demonstrate that we are the critical interface between the patient and the technology. We must be reminded that we do not perform ventilator checks, but rather patient-ventilator assessments. We do not perform nebulizer treatments, but rather patient treatments. Due to the misallocation of respiratory care services that commonly occurs, we also add value because we serve as gatekeepers.[24-26] In other words, part of our responsibility is to police the allocation of resources. We have a responsibility to our patients to interact with their physicians to assure that the most appropriate care is provided—even though that sometimes means that no respiratory care is needed. It has been demonstrated that respiratory therapists' interactions with physicians can significantly reduce the number of inappropriate respiratory care treatments.[27] Such interactions add value and lower costs! Much of the valuable role of the respiratory therapist at the bedside is to serve as a technical and clinical consultant.

Avoiding Task Orientation

Unfortunately, many respiratory therapists are task-oriented. In many hospitals, respiratory therapists visit the patient intermittently to perform a task—eg, chest physiotherapy, ventilator check, arterial puncture, or nebulizer treatment. The question is increasingly asked, Why not allow clinicians who are already present at the bedside (eg, nursing staff) to perform these tasks? I believe that we must accept the reality that some low-level tasks (eg, incentive spirometry) can be performed in a cost-effective manner by persons who are not respiratory therapists. But we need also to demonstrate that the respiratory therapist brings much more to the bedside than the ability to perform tasks. Respiratory therapy involves patient assessment and interaction with physicians to assure that the best therapies are prescribed—much more than simply performing a task. If respiratory therapy is limited to the performance of tasks, then the skills to perform those tasks can be assumed by others. The challenge to all of us is to demonstrate that respiratory care involves much more than task performance. This adds value to the role of the respiratory therapist.

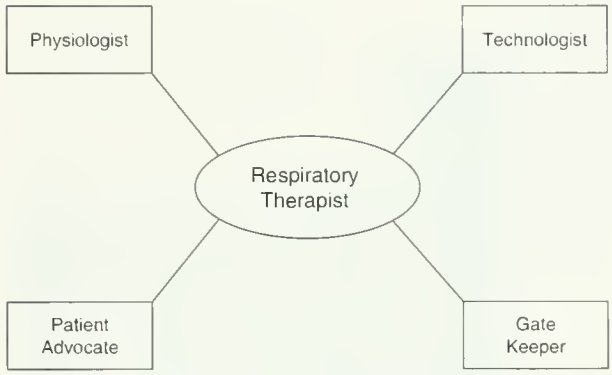

Fig. 2. The evolving role of the respiratory therapist.

It is also important to recognize that there are some tasks that are performed better by respiratory therapists than other health-care providers. Several studies have shown that respiratory therapists are more knowledgeable than physicians or nurses in the correct use of metered dose inhalers (Fig. 3).[28-30] It would, therefore, make sense for respiratory therapists to instruct patients in the use of an inhaler. Although it may be foolish to expect that all respiratory care will be administered by respiratory therapists, it is important for respiratory therapists to use their knowledge to educate others who administer respiratory care. Because of their education and training in respiratory care,[31] respiratory therapists can add value at the bedside by teaching such simple things as oxygen therapy and pulse oximetry—both commonly used but not always well understood or appropriately provided by nurses and physicians.[32-34]

Developing High-Level Skills

Although respiratory therapists should avoid a task orientation, it is important that we develop, maintain and expand the high-level tasks that we can provide. It makes little sense

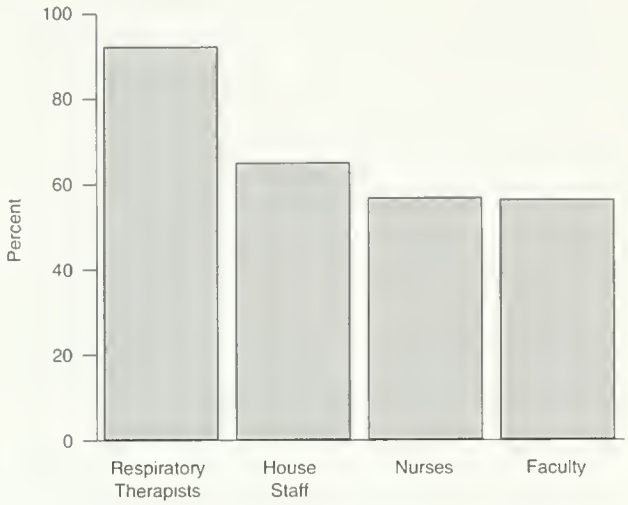

Fig. 3. Comparison of the ability of various medical personnel to correctly teach patients to use metered dose inhalers. The y-axis represents the percent of each group to correctly teach the steps involved in the use of an inhaler. Data are from Reference 29.

to have a respiratory therapist provide every incentive spirometry treatment, but it makes a lot of sense to train respiratory therapists to intubate,[35-40] insert arterial lines,[41] perform hemodynamic monitoring,[42] perform defibrillation and other resuscitation procedures,[43-48] and maintain extracorporeal life support systems.[2,49,50] Although we should not limit our value to the skills we provide, we can add value by our ability to perform high-level tasks.

Flexibility & Accepting New Roles

Those therapists who are flexible and willing to accept new roles will survive. This is not a time to say, It's not my job. It's time to say, I want to do that, I can do that, and I will do that. We must recapture the entrepreneurial spirit that made arterial blood-gas analysis and chest physiotherapy a part of respiratory therapy more than 20 years ago. We must re-engineer our thinking and our actions. The way that many of us have thought of our roles in the past is no longer effective. We must think in cost-effective, value-added, and patient-first terms. We must be change agents, and not individuals who oppose change. As a profession, we must become more involved in activities designed to avoid hospital admissions and to shorten hospital stays when they occur. This means employment in sites such as subacute care and home care.[51,52] It also means greater involvement in preventive and primary care, such as asthma education.[53-59]

Maintaining Our Focus on the Patient

Although we might not agree with some of the models used to achieve this objective,[60,61] we must all agree that patient-focused care is necessary and important. We must re-engineer our thinking to put the patient first in every clinical and administrative decision we make. Our every action should be selected with consideration of how it affects the patient. The health-care-delivery system exists because of the patients it serves. We place ourselves in a precarious position when our actions are governed by what is convenient for the therapist rather than what is best for the patient. It makes sense to reorganize the respiratory care department to assure continuity of respiratory care. This involves limiting the number of therapists who see a given patient and enhancing the therapist's skills so that the best care is rendered to meet the needs of that patient. This also places the therapist in a position to become a dynamic part of the patient-care team.

Providing Clinical Leadership

We must assume leadership in clinical issues at the bedside. We must provide the clinical leadership for respiratory care including patient assessment and development of the care plan in addition to administering the therapy. We must be more than the task provider. We must be experts on patient management strategies, such as permissive hypercapnia,[62-66] prevention of ventilator-associated acute lung injury,[67-72] and noninvasive positive-pressure ventilation.[73-76] We should be able to speak fluently about high-frequency oscillation[50,77] and extracorporeal oxygenation and carbon dioxide removal.[49,50,78] We should be at the forefront of emerging technologies such as inhaled nitric oxide,[79] liquid ventilation,[80-84] and tracheal gas insufflation.[85-87] We should be promoting patient care based upon our knowledge of the evidence (ie, scientific literature) and not because "We always do it that way" or "...because that's what the doctor ordered."[88,89]

Reducing Cost without Compromising Quality

All of us must participate in the efforts to reduce costs without compromising quality. We should be looking for ways to work smarter. We add value when we provide leadership in this area. We need to look around ourselves and question costly practices that may no longer make sense. Do we need to routinely change ventilator circuits?[90-92] Can we use passive humidification instead of heated humidifiers in mechanically ventilated patients?[93,94] How often do we need to perform patient-ventilator system checks? Can we substitute metered dose inhalers for nebulizers (Fig. 4)?[95-97] Can we use a postoperative oxygen therapy protocol to decrease the costs associated with the use of pulse oximetry and oxygen therapy (Figure 5)?[98]

Recognizing Interdependence with other Clinicians

We must become interdependent with other clinicians—particularly physicians and nurses (Fig. 6).[99] Interdependence is a characteristic of effective people.[100] Much of the deliv-

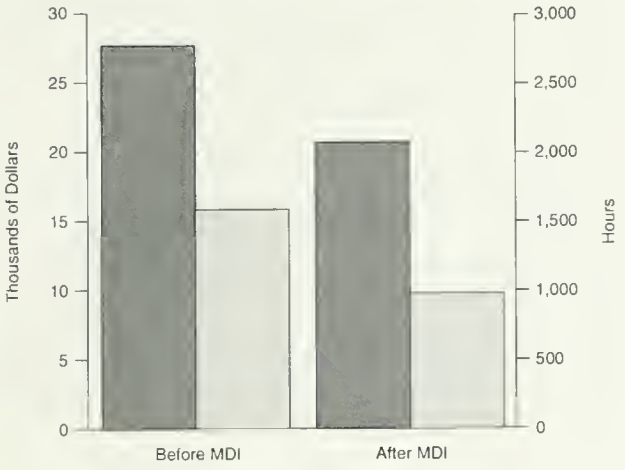

Fig. 4. Cost (■) and time (□) savings per month before and after initiation of a program to authorize respiratory therapists to substitute metered dose inhalers (MDI) for nebulizers in hospitalized patients. Data are from Reference 34.

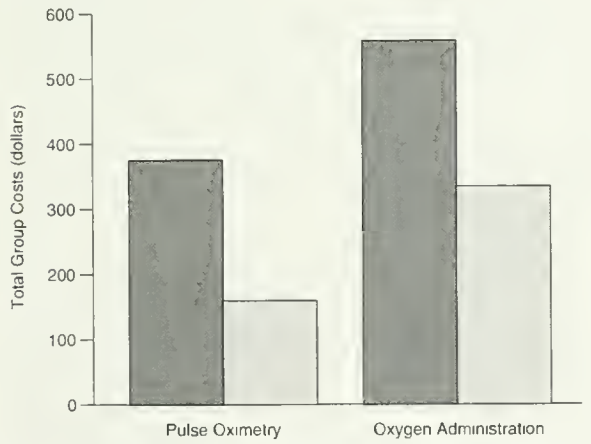

Fig. 5. Cost comparison for standard practice (Group 1, n = 20, ■) and for use of an oxygen administration protocol (Group 2, n = 20, □). Data are from Reference 98.

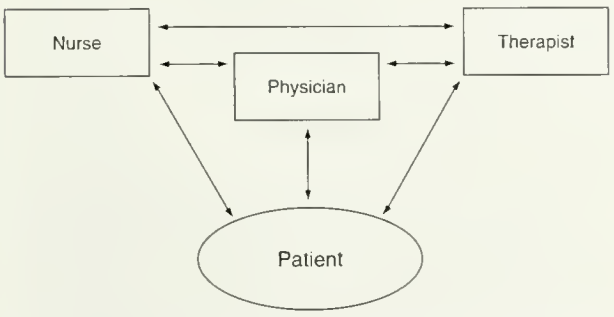

Fig. 6. The interdependence required of professional health-care providers at the bedside to deliver optimal patient care.

ery of health care is collaborative,[101-104] and we must create the expectation that we are always present and that we contribute an irreplaceable value by our presence. We cannot wait to be called—the call may never come. We must anticipate the need so that the call is never necessary. Over the past 40 years, respiratory therapists have enjoyed a very special relationship with physicians. We must continue to strengthen our relationships with physicians so that they will view us as necessary colleagues and allies at the bedside.

Adopting Valid Guidelines & Protocols

Respiratory care protocols have become the respiratory care 'buzz word' of the 1990s and have been the subject of numerous reports in this Journal[97,98,105-112] and others.[113-116] Medical staff approved protocols allow therapists to determine what care is appropriate for a patient and to modify or discontinue that care as appropriate. Protocols extend the skills of the therapist beyond providing tasks—to assessment and development of respiratory care plans. Well-written protocols are flexible guidelines that adapt to the needs of specific patients. They are more like 'assess-and-treat' or 'consult' services than strict protocols, per se. The AARC Clinical Practice Guidelines[117] have been used in many institutions to develop protocols. Because the Clinical Practice Guidelines are literature-based, using the Guidelines to develop respiratory care protocols results in protocols that are scientifically based.

Another buzz word of the 1990s is 'critical pathways'.[118] Unlike respiratory care protocols, critical pathways are designed to be multidisciplinary. Critical pathways were introduced into health care in the 1980s as a method of improving hospital efficiency. Specific goals of pathways include selecting the best practice, defining standards for hospital stay and for the use of treatments and diagnostic tests, examining the interrelationships between different aspects of the care plan to avoid redundancies, providing a common plan of care for all care providers, decreasing the amount of documentation, and improving patient satisfaction. Critical pathways are being developed in many hospitals, and respiratory therapists should be involved in the development of pathways related to respiratory care. Although critical pathways are proliferating, their impact on patient care and health-care costs remains to be determined.

Increasing Our Educational Level

The appropriate education level for respiratory therapists has been debated for many years.[119,120] With the move away from a task orientation to that of assessment and care-plan management, we must ask ourselves whether a 1-year entry level is appropriate. It seems to me that the skills necessary for respiratory therapists today and in the future require an educational program that teaches assessment and implementation of care plans in addition to skills training. I have

a strong belief that maturity develops with a liberal arts education and scientific reasoning develops with a basic sciences education. Such maturity and reasoning skills cannot be achieved in a 1-year program. Regardless of the entry-level, all of us must continue our formal education. The higher the education level of our profession, the greater will be the respect that we engender from others.

Participating in Continuing Education

We must do everything we can to expand our knowledge because knowledge is power. Expanding our knowledge means reading journals and attending educational conferences. A relatively new source of knowledge is the Internet.[121-129] The respiratory care and critical care newsgroups on the Internet are accessible to anyone with a computer and a modem and allow anyone in the world to discuss issues of interest.

We must also contribute to the scientific basis of respiratory care. Only in that way will we be viewed as true professionals. We must be fluent in the current literature affecting respiratory care and be able to discuss it with the health-care delivery team. This is another example of how respiratory therapists add value beyond their ability to perform tasks. We should read not only RESPIRATORY CARE, but other related journals such as *Chest*, *American Journal of Respiratory and Critical Care Medicine*, *Critical Care Medicine*, *Anesthesiology*, and *Pediatric Pulmonology*.

Becoming Involved in the Community

There is much we can do within the community to strengthen the image of respiratory therapists. For example, we can teach resuscitation courses, work with the American Lung Association and the American Heart Association, and help with asthma camps and asthma education programs. Even within our individual hospitals, we can volunteer to participate in professional committees throughout the hospital. All of these activities help define our professional image. Our involvement, throughout the hospital and within the community, projects the image that we are professionals who are capable of doing more than 'giving treatments.'

Developing Teamwork & Accepting Empowerment

Teamwork and empowerment are two more buzz words of the 1990s. We must learn to work together as teams of respiratory therapists (respiratory care department) and teams of health-care providers (therapists, physicians, nurses). We must make effective contributions to the health-care delivery team. Because many respiratory care departments now have fewer managers and supervisors, staff respiratory therapists are given responsibilities beyond those of the traditional staff therapist of the past—they are empowered. With this responsibility and empowerment comes accountability. This forces staff therapists to become professionals, with an expanded focus beyond the tasks that are assigned.

Supporting Our Professional Organization

We must support our professional organization (ie, the AARC). It is important for respiratory therapists to have a passion for their profession, and one way that can be demonstrated is by membership in the professional organization, both locally and nationally. Supporting the credentialing process is also important (ie, the National Board for Respiratory Care [NBRC]). Although the importance of licensure should not be underrated, the goal of every respiratory therapist should be to become a registered respiratory therapist.

Hope for the Future

I believe we can survive as a profession. However, to avoid extinction we must guard against apathy, anger, and absence. Rather than being apathetic about our role, we must aggressively defend and expand the contributions of the respiratory therapist. We must have a passion for the profession. Rather than being angry about changes in health-care delivery, we must offer positive solutions that emphasize the contributions of the respiratory therapist. We must be change agents—not defenders of the status quo. Finally, we cannot afford to be absent from the bedside. We must make a contribution at the bedside that demonstrates the value of the respiratory therapist in everything we do.

Let's join together to demonstrate to the world that respiratory therapists are a bargain—we provide high-quality care at a reasonable cost. We use 'high-tech' equipment (ventilators, extracorporeal membrane oxygenators), we perform all traditional respiratory care procedures (aerosol administration, chest physiotherapy, arterial blood-gas analysis), we perform advanced procedures (intubation, hemodynamic monitoring, arterial catheter insertion), and we implement and manage mechanical ventilation strategies (including weaning). Further, we are available 24 h/day every day of the year, and we cost less than most other licensed health-care professionals.

> ...you'd better start swimming or you'll sink like a stone, for the times they are a-changin ...
>
> Bob Dylan

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Right Paratracheal Density in an Asymptomatic Patient

Luis Teba MD and N LeRoy Lapp MD

Case Summary

A 45-year-old white man was referred to our pulmonary clinic for evaluation of a right paratracheal mass found on his chest radiograph taken during the annual health surveillance at his work place, a steel mill. The mass was reported as enlargement of the lymph nodes. Re-evaluation of chest radiographs dating back 5 years disclosed no abnormality.

The patient had no health complaints and denied cough, dyspnea, chest pain, fever, chills, fatigue, night sweats, weakness, weight or appetite changes, skin rash, or problems with vision. He also denied any history of back injury or back pain.

He had been diagnosed with non-insulin dependent diabetes mellitus 4 years earlier and was being treated with glyburide (Micronase®) 20 mg/day. He had sustained a fracture of the right clavicle with no pulmonary injury as the consequence of a motor vehicle accident 2 years prior to admission. He denied past exposure to tuberculosis and had no history of upper respiratory problems, thyroid disease, or malignancies. He did not recall previous skin testing for tuberculosis (purified protein derivative, or PPD).

The patient has been working at the steel mill for 22 years with heavy exposure to graphite and dust. He has a 25 pack-year history of cigarette smoking.

Physical examination disclosed normal vital signs and no skin lesions, clubbing, or cyanosis. The conjunctivae and lacrimal glands were normal. Palpation of the neck revealed no enlargement of the thyroid or lymph nodes. Examination of the chest demonstrated a moderate protuberance at the middle of the right clavicle. Chest excursion was normal, and auscultation revealed no stridor, wheezes, or crackles. No cranial nerve deficits or muscle weakness could be detected. Laboratory studies revealed a hemoglobin level of 14.3 g/dL, a white blood cell count of 7.3×10^9/L with normal differential, and platelet count 199×10^9/L.

Skin tests with PPD, candida, and mumps antigens were nonreactive. Posteroanterior (PA) and lateral chest radiographs are shown in Figure 1.

Dr Teba is Professor of Medicine & Anesthesiology and Dr Lapp is Professor of Medicine, West Virginia University Health Sciences Center, Department of Medicine, Section of Pulmonary and Critical Care Medicine, Morgantown, West Virginia.

Reprints: Luis Teba MD, Department of Medicine, Section of Pulmonary & Critical Care Medicine, West Virginia University HSC, Morgantown WV 26506.

How would you answer these questions?

What abnormalities are present in Figure 1? _____

What diagnoses should be considered, given the history, physical exam, and laboratory and radiographic findings? _____

What other diagnostic procedures do you suggest? _____

Answers and Discussion on Page 546

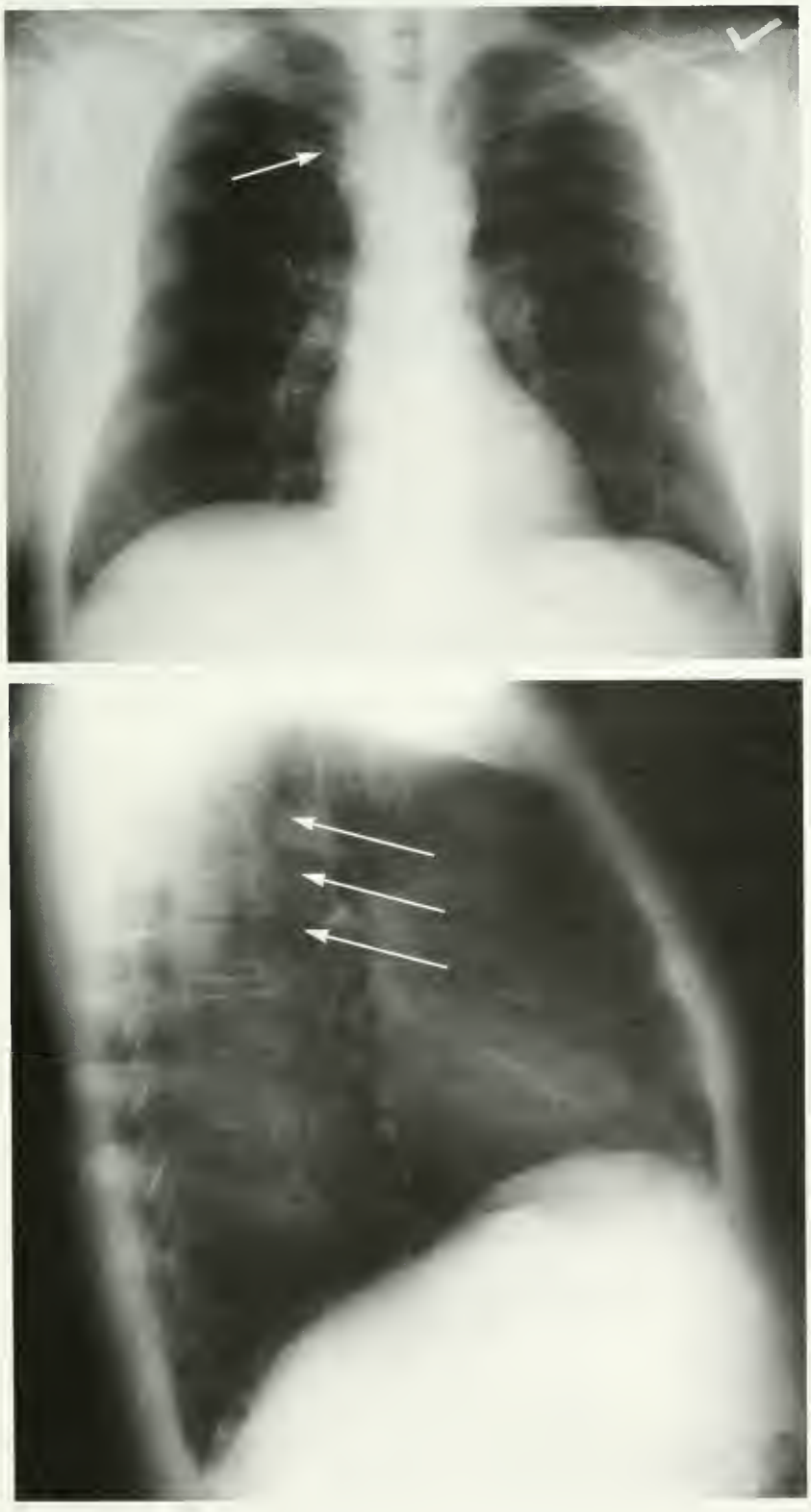

Fig. 1. Chest radiograph showing enlargement of the paratracheal area and degenerative changes in the dorsal spine (arrows).

Radiographic Findings. PA chest radiographs show enlargement in the right paratracheal area. The lung parenchyma is free of masses or infiltrate. The lateral view reveals degenerative changes in the mid-to-lower dorsal spine.

Differential Diagnosis. Abnormalities of any anatomic structures of the mediastinum may present as a mediastinal density on the chest radiograph.[1] The configuration of the mediastinum can be affected by many disorders, many of which are benign processes that originate in the mediastinum, whereas others are metastatic lesions.[2] Mediastinal abnormalities may be found incidentally on conventional chest films (ie, plain films). In the asymptomatic patient, many of these disorders are benign; however, approximately half of symptom-causing mediastinal densities are due to malignant processes. Symptoms arising from mediastinal masses are usually due to compression or invasion of adjacent intrathoracic structures. Cough, dyspnea, dysphagia, and chest pain are the most frequent manifestations.[3] In rare instances, a paraneoplastic syndrome may be observed in the patient with a mediastinal tumor.[4]

Tumors of the mediastinum have long been grouped according to the anatomic location into either the anterior, middle, or posterior compartment. The location of a density in the anterior compartment gives rise to the possibility of its being caused by one of the so-called LT conditions (ie, lymphomas, teratomas, thymomas, and thyroid tumor). In addition, benign and malignant vascular tumors, including cavernous hemangiomas can also distort the normal mediastinal configuration.[5]

The heart, the trachea, the pulmonary hila, and important groups of lymph nodes are located in the middle mediastinal compartment. Pathologic processes affecting these structures could appear as mediastinal masses on plain chest films.

Neural tumors and pathologic processes of the esophagus, descending aorta, paravertebral lymph nodes, and vertebral bodies are some of the conditions found in the posterior mediastinum that could potentially be seen as paratracheal densities on the chest radiograph.

Occupational exposure to graphite and other 'dust,' important in our patient's history, can cause mediastinal changes similar to those seen in silicosis and coal workers' pneumoconiosis (CWP). However, by the time enlarged mediastinal nodes are observed parenchymal pulmonary changes are usually already present.[6]

Course of Action. Testing revealed that the patient's serum angiotensin converting enzyme (ACE) level was 83 U/L (normal 20-60 U/L). A computerized tomography of the thorax was obtained (Fig. 2). It reveals osteophytes, or 'spurs,' that extend along the thoracic spine and are most prominent in the upper-right thoracic spine. No paratracheal adenopathy, pulmonary nodules, or mediastinal masses are seen.

Diagnosis. Diagnosis of diffuse idiopathic skeletal hyperostosis was established. Paratracheal projection of the vertebral spur was the cause for the "paratracheal density" observed on the plain chest radiograph.

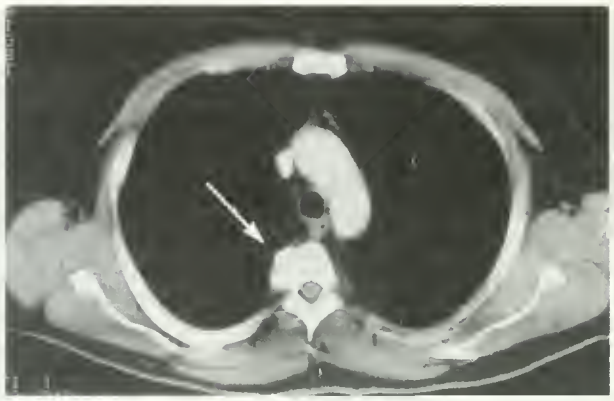

Fig. 2. A 'foot-up' computerized tomogram revealing osteophyte formation in the spine (arrow).

Discussion

The presence of a unilateral paratracheal density should alert one to the possibility of conditions including tuberculosis, fungal infections, lymphoma, bronchogenic carcinoma, and metastatic disease. Although not typical, isolated right paratracheal adenopathy has been described in some cases of sarcoidosis.[7,8]

Diffuse idiopathic skeletal hyperostosis (DISH), characterized by marked osteophytosis and spur formation, has been described under different names.[9,10] DISH affects the vertebral column most commonly, with the initial involvement by spur formation of the anterior and lateral aspect. The right side of the thoracic spine is involved more often than the left. In patients with long-standing disease, extensive extraspinal involvement may also be seen. DISH is usually reported in middle-aged and elderly persons, with a male preponderance. The majority of patients are asymptomatic. There are no specific or consistent laboratory abnormalities and etiology and pathogenesis of DISH is still unknown. A strong association of this disease with diabetes mellitus has been established, and abnormal glucose tolerance has been reported in a high percentage (up to 40%) of patients with DISH.[11,12] Hyperostosis is also commonly seen in patients with sickle-cell disease and may involve the vertebral bodies causing a "DISH-like" syndrome in some patients.[13] In patients with DISH, the anterolateral spur formation may appear on the PA chest radiograph as a bilateral or unilateral paratracheal lymphadenopathy as it was reported in our patient.

Sarcoidosis is a multisystem granulomatous disorder of unknown etiology.[14] A significant percentage of patients with sarcoidosis are asymptomatic, with the disease being detected during radiographic screening for other unrelated purposes. Although sarcoidosis presents most frequently with bilateral

symmetrical enlargement of the hilar nodes, the presence of right paratracheal adenopathy has been described in some series.[7,8] Angiotensin-converting enzyme (ACE) is usually increased in sarcoidosis, however, ACE elevation has also been reported in other conditions mimicking sarcoidosis and in diabetes mellitus.[15]

In our patient, the right paratracheal shadowing seen on the PA chest radiograph was mistaken for right paratracheal-lymph-node enlargement. In the absence of symptoms, the elevation of the serum level of ACE and the absence of skin testing reactivity were initially suspected to represent sarcoidosis. Later the radiographic findings were explained by the vertebral hyperostosis and the mild increase in ACE from his diabetes.

Diagnostic workup of mediastinal abnormalities may require tissue biopsy; however, before one rushes to surgical excision and tissue diagnosis, the patient's overall clinical condition should be evaluated. A search for extrathoracic lymph nodes or other extrathoracic manifestation of the process may provide sufficient information to establish the etiology of the mediastinal abnormality. In addition, examination of old radiographs, when available, may demonstrate that the mediastinal anomaly in question is old and not progressing. Careful assessment of lateral views may disclose the location and nature of the mediastinal density seen on plain radiographs.

If, despite the described approach, the etiology of the mediastinal density is still uncertain, then diagnostic workup should proceed with computed tomographic (CT) imaging with contrast or magnetic resonance imaging. These techniques can supply additional information about mass size, location, density, homogeneity, and involvement of contiguous structures. Furthermore, some recent publications suggest that PET (positron emission tomography) imaging may be an excellent tool for determining whether intrathoracic lesions are malignant in origin.[16]

In order to establish a definitive diagnosis, histologic examination of tissues from the lesion may still be required in a large number of patients with mediastinal densities. In many instances, mediastinal tissue can be obtained by CT or bronchoscopy-guided needle biopsy, mediastinoscopy, and pleuroscopy, without the need for open thoracotomy.

We suggest that diffuse idiopathic skeletal hyperostosis always be considered in the differential diagnosis of apparent paratracheal shadowing. If we had assumed that the patient had sarcoidosis and had not opted to pursue a CT study of the chest, the real nature of the radiographic abnormality would not have been established. The unresolved diagnosis would have required serial follow-up radiographic examinations, perhaps biopsy, and a patient living in fear of sarcoidosis.

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Entry-Level Exam Review for Respiratory Care: Guidelines for Success, by WV Wojciechowski. Illustrated, 332 pages. Albany, NY: Delmar, 1995. $31.50. **Advanced Practitioner Exam Review for Respiratory Care: Guidelines for Success,** by WV Wojciechowski. Illustrated, 475 pages. Albany, NY: Delmar, 1995. $39.95.

Success at the National Board for Respiratory Care's (NBRC) credentialing examinations has proved to be an ongoing challenge for students and educators alike. For the student dealing with a demanding academic and clinical schedule plus work and family demands, preparing for the exams can be a stressful experience. For the educator who must deal with a continually growing body of knowledge and pressure to decrease numbers of credit hours, preparing students for the examinations as well as imparting other important knowledge not covered on the tests (ie, interpersonal skills, ethics, critical thinking) can be a daunting effort. The instant review manuals by Wojciechowski provide a useful adjunct for students preparing for the entry-level and written, advanced-practitioner examinations.

Each text is similar in format. Each begins with an explanation of how to use the text. Included in this section is background information about the NBRC's testing methods, a reproduction of the relevant 1993 examination matrix, and a description of item types. The introductory section also has a discussion of the technique for taking multiple-choice and true-false examinations. This particular piece would be useful to students just beginning their respiratory care training because they will, no doubt, be taking tests with these formats.

Following the introduction is a chapter on test preparation. Several important topics in addition to the actual preparation for the examinations are explored. The details of the application procedure are reinforced; and suggestions for activities the day before and the day of the examination are shared in the entry-level text.

With the exception of brief appendices (quick reference clinical data in the entry-level and advanced-practitioner texts, and waveform interpretation in the advanced-practitioner text), the remainder of the texts is made up of sample test items. Each sample item (645 in the entry-level examination and 750 in the advanced-practitioner test) is accompanied by its own detailed analysis and content source references. Each item is also referenced to its place in the NBRC examination matrix, allowing test candidates to identify weak areas for more intensive study. All areas of the NBRC exam matrices are well represented in the sample items.

The test items are divided into 5 sections. There is a pretest and posttest with the same number of items to be encountered in the actual examinations (140 for the entry-level and 100 for the advanced-practitioner examinations). Between the pretest and posttest are 3 sections corresponding to the 3 major sections of the NBRC matrices: clinical data, equipment, and therapeutic procedures—each section containing 105 to 212 items. As an educator, I can verify that the more sample or study questions I provide my students, the better they like it. The number of questions provided in these texts should satisfy even the most demanding "question junkie."

These review texts represent a useful addition to any test candidate's study arsenal. They are easy read and contain a wealth of sample items and a liberal number of high-quality figures, diagrams, and tables. I recommend these texts to anyone preparing for the entry-level or advanced-practitioner respiratory care examinations.

Randy De Kler MS RRT
Cardiopulmonary Care Sciences
Department
Georgia State University
Atlanta, Georgia

Equipment Theory for Respiratory Care, Second Edition, by GC White MEd RRT CPFT. Hardcover, 666 pages, illustrated. Albany, NY: Delmar Publishers, 1996. $53.95.

A solid understanding of respiratory care equipment is essential to successful practice. This book is intended to guide respiratory care students to a thorough understanding of this equipment. White, who is director of Clinical Education at Spokane Community College in Spokane, Washington, shows an understanding of the detail students need when first learning new concepts. In his instructions to the student, White gives some good advice by suggesting that students read the introduction and think about what it means before proceeding. The next step is to read the objectives and to rewrite them as questions to be answered as the text is read. Readers are encouraged to read the text and view the text, artwork, and diagrams together to see the whole presentation. Then, students should follow up with laboratory work with the equipment. Finally, he suggests rereading the objectives and taking the self-assessment quizzes. If further information is required, he refers students to the bibliography.

The book is relatively easy to read with language as simple as the topic permits. Chapter layout includes an introduction, objectives, text, and a self-assessment quiz. The quizzes seem complete and straightforward. Whenever formulas are presented, an example is also given. The diagrams are crisp and easy to understand. For example, in the first chapter, White explains how diverging and converging ducts differ in their effect on gas flow. Because the concept is shown visually, it is easy to understand.

Most chapters begin with a review of the physics principles used in that chapter. I think this will help students relate specific equipment function to general principles. The review is followed with descriptions of the equipment covered. For each piece of equipment discussed, a detailed description of assembly and troubleshooting, in a separate box, is presented followed by a troubleshooting algorithm. For an experienced practitioner, the algorithms may appear overly detailed and cumbersome. To the inexperienced student, however, they provide a guide through the thought processes necessary to solve the problems of the sometimes complex tools of our trade. For example, the algorithm for determining if an oxygen delivery device is working seems overly complex. On the other hand, the flow charts for the intermittent positive-pressure breathing (IPPB) machines looks useful. These algorithms do have the potential for teaching problem-solving and troubleshooting skills to the uninitiated. I am concerned, however, that students reared on these algorithms may be unable to

troubleshoot a new piece of equipment without them. These extensive troubleshooting sections also interrupt the flow of the text and make reading a little disjointed. I might have placed these in an appendix at the back of the book or at the end of the chapter.

Content is fairly representative and complete. Chapter 1 thoroughly covers gas physics, medical gas equipment, and gas delivery equipment—including piping systems, cylinders, and patient equipment. A separate list of safety rules for oxygen cylinders is easy to read and understand. Also included is equipment such as oxygen concentrators, demand flow devices, liquid oxygen systems, oxygen reservoir systems and the transtracheal oxygen catheter used in the hospital and in home care.

Chapter 2 covers humidity and aerosol therapy equipment. The chapter begins with a discussion of the physics and principles involved in humidity and aerosol therapy. This includes humidity measurement, evaporation, humidity deficit, the physical characteristics of aerosols and particles, and factors that influence aerosol deposition in airways. This is followed by a review of several different kinds of humidifiers, such as the bubbler, pass-over, jet, and Cascade. Heat and moisture exchangers are also included. The chapter concludes with a review of nebulizers including both small volume nebulizers designed for medication delivery, metered dose inhalers (MDIs), spacing devices for MDIs, and large volume nebulizers.

Chapter 3 discusses incentive spirometry and IPPB equipment. White includes a brief review of the physics involved in volume measurement and gas pressure and flow regulation. He then reviews the function of several brands of incentive spirometers and the prominent or often used IPPB machines: Bird Mark 7, 7A, and 8; and the Puritan Bennett PR-2 and AP5. The chapter ends with a review of intrapulmonary percussive ventilation and other chest percussors.

Emergency resuscitation equipment is covered in Chapter 4. After a review of the physics involved, specific artificial airways, manual resuscitators, cuff-pressure meters and suction devices are described. The diagrams in this chapter are clear, especially those showing the manual resuscitators. There are also diagrams demonstrating proper placement of the oral and nasopharyngeal airways and the endotracheal tube.

Chapter 5 is devoted to physiologic measurement and monitoring devices. Included in this chapter are pulmonary measurement devices from water-seal spirometers to the body plethysmograph and everything in between. This is followed by a description of oxygen analyzers, blood gas electrodes, transcutaneous devices, and oxygen saturation measurement. The chapter ends with a section on end-tidal CO_2 monitoring.

Mechanical ventilation is discussed in Chapter 6, beginning with the physics and classification system of ventilators. White has opted to use Chatburn's classification system.[1] I find this system relatively straightforward and easy to understand. White discusses each variable separately in this chapter and includes a chart comparing various breathing patterns, such as controlled manual ventilation, assist/control, intermittent manual ventilation, synchronized intermittent mandatory ventilation and continuous positive airway pressure, in terms of how mandatory and spontaneous breaths are initiated and controlled. I think this chart would be helpful to students trying to understand the differences in various modes.

Chapters 7 through 10 focus on specific ventilators. Each ventilator is presented, again, according to Chatburn's classification system. In Chapter 7, the adult acute care ventilators using volume as a control variable are discussed. Chapter 8 presents adult acute care ventilators that use flow and pressure as control variables. Chapter 9 discusses pediatric and neonatal ventilators, and Chapter 10 introduces home care and transport ventilators.

This equipment book is as complete as any I have seen in the last few years. It is easy to use and comprehensive enough to cover at least 2 and, possibly 3 semesters in the average respiratory care program. This is important to students because it reduces book costs. The book has features such as objectives and a postquiz to assist the student in the learning process. I think this book serves its intended purpose well.

Gretchen Kenner Riehl MS RRT
Clinical Coordinator
Respiratory Care Program
El Centro College
Dallas, Texas

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Shortness of Breath: A Guide to Better Living & Breathing, 5th edition, by AL Ries, KM Moser, PJ Bullock, TM Limberg, R Myers, DE Sassi-Dambron, JB Sheldon. Hardcover, 114 pages, illustrated. St Louis: Mosby, 1995. $9.95.

This is a self-help book written to educate patients with chronic lung disease especially aimed at patient's with chronic obstructive pulmonary disease (COPD). It is written with the intent of educating a lay patient population. From this point of view, it certainly meets its goals.

There are numerous self-help books on the market today many of which are aimed at the patient with asthma. Several of those books have previously been reviewed in RESPIRATORY CARE. Such books have been written by celebrities, self-proclaimed experts, and seasoned professionals. This book has multiple authors with a wide range of professional backgrounds and credentials. As this is the fifth edition of the book, it has certainly found its niche in the marketplace. The book's 19 chapters explain the causes of shortness of breath, how the lungs work, characteristics of various lung diseases, how various medications, oxygen, and breathing equipment work, and how to live a cleaner, happier and healthier life.

It is important for the health-care professional to know about these books because many of our patients may be reading them and we may be called upon to comment on their content. Of equal importance is the ability of the respiratory care professional to recommend to patients specific books as being superior.

The book is written on a simple level. In some cases, the simplicity is so great as to cause some inaccuracies. As an example, the book refers to oxygen as an "Energy Source." The chapter about COPD is well written, defines other illnesses, and distinguishes chronic bronchitis from emphysema and asthma. Although it may have been beyond the scope of the book, I would have liked to see mention of the National Heart, Lung, and Blood Institute's Guidelines on the treatment of asthma, if only as a resource some patients may wish to consult. The chapter about medication explains the difference between generics and branded pharmaceuticals, and the chapter about oxygen and aerosols provides valuable information on oxygen safety as well as good instruction on the use of metered

dose inhalers (MDIs). It also mentions spacers but does not provide adequate information on how to use them. Tips on exercise, diet, relaxation techniques, and better approaches to day-to-day living are also given.

Cartoon-like illustrations help provide visual instructions in every chapter. Some examples are a cartoon showing the technique of pursed lip breathing, and one showing the technique of diaphragmatic breathing. The flotation technique for determining when an MDI is empty, a common question from patients, is also illustrated.

This is a good book for patients recently diagnosed with COPD and may also be useful to long-term patients seeking education about their disease or as a refresher of previously learned information. Many professionals will find this book too simplistic. It may be useful for them to know of its existence for recommendation to patients. Occasionally, an analogy used in this book may be adopted by some professionals for their own use when teaching patients.

Philip L Schiffman MD FACP FCCP
Diplomate, Pulmonary, Critical Care, & Internal Medicine
Pulmonary & Intensive Care Specialists of New Jersey
East Brunswick, New Jersey

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Letters

LIFECARE Coordinator Objects to WOB Paper

After reading the article by Branson and Davis[1] "Work of Breathing Imposed by Five Ventilators Used for Long-Term Support ...," I have several issues to raise. As LIFECARE's Product Coordinator for the PLV-102, I have a keen interest in this topic.

Besides technical information that is in error the article contains broad statements of opinion. An example of one such statement is, "The TBird VS and 7200ae ventilators are superior to the home-care ventilators and have a low work of breathing." While the two mentioned ventilators do have low work of breathing, they are not superior to home-care ventilators in many situations. At publication, the TBird VS was not available for purchase, so its actual patient performance is not yet known. And, the 7200ae would have trouble keeping up with a portable ventilator out in a parking lot or on a soccer field behind a wheelchair, even with a very long extension cord.

Another sweeping statement is "Weaning patients in the subacute-care environment is a concern." Yes, it is. And, more importantly, how to wean patients is a concern as well. Historically[2] and again more recently,[3] T-piece weaning has been recommended as a better technique than weaning with either pressure support ventilation (PSV) or synchronized intermittent mandatory ventilation (SIMV). This would certainly obviate issues of spontaneous breathing in SIMV.

The authors seem to suggest the use of the TBird VS in place of complicated intensive care unit ventilators for long-term care. But the goal for manufacturers is to produce ventilators for long-term care that are even less complicated than current portable volume ventilators. Of more concern is that the PLV-102 was not set up for SIMV as recommended by the manufacturer.[4] Kacmarek et al,[5] showed that the use of an H-valve markedly reduces inspiratory work of breathing to 0.009-0.053 J/L with pass-over humidification at flows from 20-60 L/min. LIFECARE aggressively teaches that H-valves should be used to decrease inspiratory WOB for spontaneous breaths in SIMV.

Many PLV-102s are in use in subacute care facilities and are very successfully ventilating patients in control, assist/control, and SIMV modes. They are also used in home care, especially with pediatric patients requiring PEEP, SIMV with continuous flow, and supplemental oxygen. Clinicians and caregivers in these situations find the PLV-102 easy to work with, choosing it for its simplicity and reliability.

There are major inaccuracies in the PLV-102 product description and specifications. The PLV-102 is microprocessor controlled, not electronically controlled. It provides the SIMV mode, delivering machine breaths synchronized with patient inspiratory effort and allowing spontaneous breaths at oxygen concentrations above 21%. Maximum trigger sensitivity is −0.2 to −0.3 cm H_2O. Volume range is 50–3,000 mL and delivered oxygen fraction can range from 0.21 to 0.90. LIFECARE's company name and address are incorrect.

In summary, I am concerned with both technical information and statements within this article. I encourage authors to be diligent in assuring accuracy and using scientific deduction in technical articles.

Sarah E Riley MPA RRT
Product Coordinator
Portable Volume Ventilators
LIFECARE International Inc
Westminster, Colorado

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Mr Branson responds:

Ms Riley takes issue with many of the points in our paper.[1] I will respond to those in the order that she presented them.

"Broad statements of opinion"—The title of our paper,[1] "Work of Breathing Imposed by Five Ventilators Used for Long-Term Support: The Effects of PEEP and Simulated Patient Demand," reveals that we compared the imposed work of breathing between devices. This being the case, the statement, "The T-Bird VS and 7200ae ventilators are superior to the home care ventilators and have a low work of breathing" is true. We did not compare color or ability to be used in a parking lot. It is interesting to note that, whereas the LP series of ventilators has been modified based on the findings of Kacmarek et al,[2] it is not clear that the PLV-102 has been improved in this regard.

"How to wean patients..."—Ms Riley's comments suggesting T-piece weaning as the preferred method of weaning are somewhat oversimplified and, perhaps, incomplete. Brochard et al[3] published a similar study looking at sicker patients who required longer periods of ventilatory support and found that pressure support ventilation was superior to both T-piece and IMV weaning.(The length of stay on ventilators in the Esteban study[4] cited by Ms Riley was less than a week.) How should the finding of increased work load on inspiratory muscles that may contribute to fatigue affect weaning choices? How does this information fit into the crafting of a rational approach to weaning long-term patients?

"Less complicated ... portable volume ventilators"—With respect to the use of simpler ventilators in long-term care, we agree with Ms Riley. However, if she will return to our paper she will note that our concern is related to the burgeoning subacute care industry. In this setting, patients with serious respiratory pathology are requiring sophisticated ventilatory management.

We agree that the H-valve system reduces imposed work. It is also an external modification requiring additional gas sources that increase the number of potential ventilator disconnections and possibly requires the use of a blender—thus increasing complexity and cost. Such problems seem contradictory to Ms Riley's earlier comments. Further, if for purposes of a bench or clinical study we modified the PLV-102, wouldn't other manufacturers cry "Foul!"? What if Puritan-Bennett asked us to control the airway pressure at the distal tip of the endotracheal tube, effectively eliminating tube resistance? The PLV-102 comes out of the box with a setting for SIMV. If it isn't capable of providing a mode without external modification, why does the labeling remain?

"PLV-102s in use in subacute facilities"—I believe that some patients in subacute care facilities today could not tolerate the imposed work of breathing created by the PLV-102. I do agree, however, that stable patients and those requiring long-term custodial care are better served by a simpler, less expensive ventilator. Those patients did not provide the impetus for our study. In fact, we think that market is well served. It is those sicker and sicker patients landing in subacute care as a consequence of health-care reform who are underserved. As stated in our paper, those patients must use an ICU ventilator with all the associated cost, size, and complexity or risk imposed work that precludes weaning.

"Major inaccuracies...in description and specifications"—We apologize for the errors related to the technical description of the PLV-102.

The main issue remains that in the face of an increased work of breathing, the PLV-102 is an inappropriate choice for sick patients in subacute care.

It is important that investigator-clinicians continue to make recommendations based on the results of scientific inquiry.

Richard D Branson RRT
Assistant Professor
Department of Surgery
University of Cincinnati
Medical Center
Cincinnati, Ohio

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Editor's Note: The 1995 Buyer's Guide Company Directory (AARC Times 1995;19(8):54) provides a more correct address for the manufacturer of the PLV-102. The 1996 Guide will appear in the July 1996 issue of *AARC Times*.

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RETROSPECTROSCOPE

Missed Opportunities

President Johnson once suggested that many important scientific discoveries were locked up in scientific laboratories and that the time had come to unlock these. I don't believe for a minute that LBJ was right. Scientists might try to publish *prematurely* (and sometimes succeed) but it's rare to find one who locks up his discoveries and keeps them hidden from the scientific public. Normal behavior of scientists is to make sure their observations and measurements are correct and then try to get them published as soon as humanly possible in the most widely read and prestigious journal in the field.

It is true, however, that researchers occasionally make important observations but don't know they've made them, or have all the information needed for a major discovery but don't make it. There's no way of telling how often it's happened and to whom but my guess is that it's an occurrence that few confess to; maybe more would after a few drinks, but rarely in writing. I call them "missed opportunities."

Sulfanilamide

Probably the most important miss here was the two-time synthesis of sulfanilamide before it was discovered a third time and finally put to clinical use. Sulfanilamide was first prepared in Vienna in 1908 by Paul Gelmo, then working for his doctoral thesis. At about the same time, Heinrich Hörlein (later to become director of the medical division of the great I. G. Farbenindustrie) went to work with the Bayer Works and centered his research work on dyes. Because sulfanilamide was an easy starting point on which to construct dyes that were particularly color-fast (presumably because of the tight combination of the sulfanilamide element with the proteins of wool and silk), he began with several dyes formed with sulfanilamide as the base. Hörlein never tested sulfanilamide for antibacterial properties, although Paul Ehrlich, a fellow German, had discovered arsphenamine, the "magic bullet" against syphilis, in 1909.

The second synthesis of sulfanilamide was in 1915, by Jacobs and Heidelberger at the Rockefeller Institute for Medical Research in New York. They decided to synthesize and test a number of chemical agents in the hope of finding one that was more bactericidal or less toxic against pneumococcal and streptococcal infections than optochin (an early but now-forgotten bactericidal drug). In their systematic search they prepared para-aminobenzene sulfonamide (sulfanilamide), much as Gelmo had in 1908. In line with then-current dogma, they thought that in order to fight infection in vivo, a substance had to be directly lethal to bacteria, and they didn't think that a substance as chemically simple as sulfanilamide could kill bacteria directly.

Heidelberger wrote in 1972 (1):

> Like everyone else at the time, Walter Jacobs and I thought that a substance had to be directly bacterial in order to be useful in combating bacterial infections. We had been successful with trypanosomiasis by applying Jacobs' idea of changing the –OH of –COOH to NH_2, as $-CONH_2$, in order to get an organic arsenical past tissue barriers. Accordingly, when we tried to get something better than optochin against pneumococcal and streptococcal infections, we started first with amides and then, by analogy, went on to $-SO_2NH_2$. *The possibility that any substance as simple as sulfanilamide could cure bacterial infections never entered our heads, nor did our microbiologist even ask to test it* [italics added]. We even improved Gelmo's method of preparation and went on to convert sulfanilamide into highly bactericidal substances which killed infected mice faster than the infections alone!
>
> As slaves to an idea, we missed the boat in 1915, losing the chance to save many thousands of lives and the development of the sulfonamides was delayed twenty years.

The third discovery of sulfanilamide came in 1932 when Klarer and Mietzsch, chemists at I. G. Farbenindustrie, synthesized a red dye, "Prontosil," which three years later, Domagk, a German who won the 1939 Nobel Prize, showed to be a remarkable antibacterial agent in man. However, Fourneau's group at the Pasteur Institute in Paris quickly discovered that the active component of the red dye was none other than sulfanilamide first used by Hörlein at I. G. Far-

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benindustrie in 1909, and that the rest of the compound was totally unnecessary.

No one will ever know whether chemists and pharmacologists at I. G. Farbenindustrie in the 1930s synthesized and tested *sulfanilamide* first or whether they tested only Prontosil and not sulfanilamide. The facts are (1) that Hörlein used and patented sulfanilamide in 1909 (2a) and that by the 1930s it was no longer repatentable, and (2) that although Klarer and Mietzsch prepared what in 1932 proved to be a powerful antibacterial agent in mice, Domagk did not publish clinical data until 1935. The suspicion will always linger that I. G. Farbenindustrie re-discovered and tested sulfanilamide first and spent the next few years in the early 1930s camouflaging ordinary sulfa into a new, complex, and above all, patentable compound—the red dye, Prontosil. Hörlein, who was connected with the story from beginning to end, never told any more than he wrote in 1935 (2b):

> Further work on the azo-compounds by our chemists Mietzsch and Klarer led to the discovery of bodies with an incomparably greater action on bacteria than that possessed by any of the azo-compounds mentioned. But even these new compounds had no effect on mice infected with bacteria. In the course of our investigations, however, Domagk observed a certain activity on the part of azo-compounds containing sulphonamide in the streptococcal sepsis of mice, thus furnishing an important starting point for the preparation of new experimental series.
>
> Azo-dyes with sulphonamide and substituted sulphonamide groups were first prepared by me in collaboration with Dressel and Kothe, twenty-five years ago. At that time we were engaged in elaborating dyes for textile purposes which, in the direct dyeing of wools, would possess a greater degree of fastness to washing and fulling than dyes free from sulphonamide, while possessing the same degree of fastness to light as the later dyes; i.e., dyes which would enter into a more intimate combination with the protein-cells of the wool than the dyes free from sulphonamide.
>
> The observation of Domagk on the action of one of these dyes on streptococci directed our subsequent work into a new channel. Numerous new azo-dye-containing sulphonamides were prepared, but the test object was no longer the wool fiber but the mouse infected with streptococci.

Artificial Kidney

This is a special case of interrelated "missed opportunities." Abel, Rowntree, and Turner in 1914 (3) published their experiments on vividiffusion in living animals but neither they nor anyone else seriously and successfully applied it to man until 1943 when Kolff did it in German-occupied Holland (4). You will say that no one could even try it in man until nontoxic anticoagulants and chemotherapeutic agents were available. But Abel and associates in 1913-14 used hirudin (that they themselves prepared from leeches), McLean published his discovery of heparin 1916, and Best (5) noted that Schmidt had prepared a material similar to heparin in 1892 (6) and Doyon again in 1912 (7).

The point with respect to anticoagulants is that earlier no one saw a necessity for purifying heparin or hirudin to permit the artificial kidney to be used in man or, if clinicians saw a critical need, they lacked chemical expertise to do it themselves and had no chemical colleagues or pharmaceutical research laboratories to turn to. Charles Best, in retrospect, had no explanation of the failure to purify heparin for use in vividiffusion but did note that "after heparin became available many people stated that they had been thinking for many years of its use in cardiac and vascular surgery" (8).

The antibacterial agents needed could have been available in 1910 or 1917 had it not been for missed opportunities in this field (see above); and antibiotics could have been available in 1930 if Fleming had taken his observations a step further (from agar plates to experimentally infected mice)—another missed opportunity.

The Carotid Sinus and Reflex Control of the Circulation

Of all experiments in the physiology student laboratory, the one most certain to produce dramatic effects is the demonstration of the carotid sinus reflex. One can clamp both common arteries below their bifurcation and see an immediate rise in blood pressure (which does not occur if the branches are clamped *above* the bifurcation) or one can increase pressure in the extracranial carotid arteries and note immediate slowing of the heart and fall in blood pressure.

These observations date back at least to 1836 when Astley Cooper noted that occlusion of the common carotid arteries led to an increase in blood pressure; he attributed it to the effects of intracranial ischemia (9). At least five physiologists between 1838 and 1885 confirmed his observations and agreed with his explanation.

Now it is important to note that in 1893 Bayliss (10) showed that these cardiovascular changes could not be due to cerebral ischemia because occlusion of both common carotid arteries did not markedly change blood flow to the medulla oblongata; the vertebral arteries still provided adequate blood flow to this region. Leonard Hill also presented strong evidence against the cerebral ischemia explanation when, in 1896, he was able to ligate successively both common carotids and both vertebral arteries in dogs without causing any abnormal behavior in the animal on recovery from these operations (11); this was because blood still flowed through the fifth artery to the brain, the anterior spinal artery. Nevertheless, Porter and Pratt still reported in 1908 (12) that raising blood pressure in the carotid arteries caused slowing of the heart by affecting blood pressure in the medullary centers. And in the same year, Eyster

and Hooker (13) artificially raised blood pressure in the carotid arteries to 200 mm Hg and recorded a marked bradycardia and hypotension (see figure 1a); this they attributed to "a direct effect of the increased blood pressure upon the cardioinhibitory centre" in the medulla. Note the strong resemblance to figure 1b taken from a paper by Hering who in 1923 at last discovered the carotid sinus, its nerve, and the effect of stimulating the nerve endings electrically or mechanically (14).

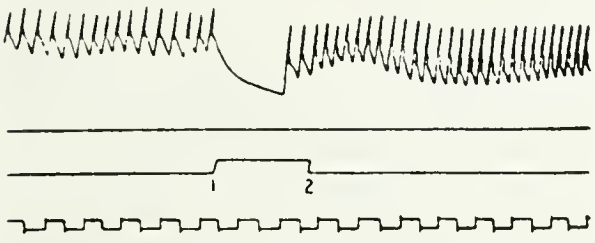

Fig. 1A. Between 1 and 2, Eyster and Hooker raised blood pressure to 200 mm Hg in the common carotid arteries of a dog while they recorded a decrease in blood pressure and heart rate from another artery.

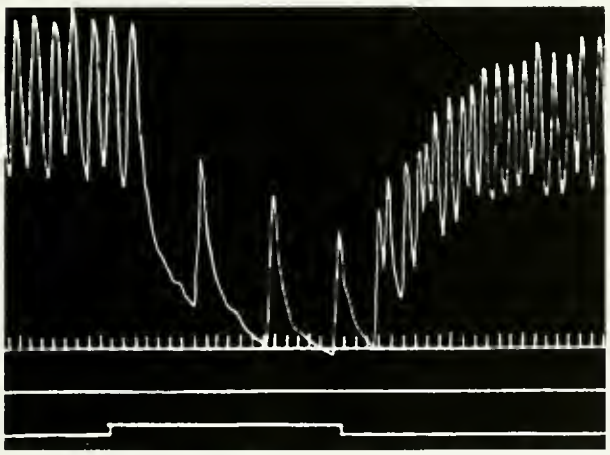

Fig. 1B. During the period indicated by the signal marker (bottom line), Hering stimulated the right carotid sinus nerve of a dog; the tracing shows an immediate fall in systemic arterial blood pressure and bradycardia.

It has been said: "We see what we look for and we look for only what we know." Eyster and Hooker had all the information needed to find the carotid sinus in 1908. All they had to do was to look an inch higher in the neck of a living dog and see the bulging, pulsating origin of the internal carotids, literally enmeshed in nerve fibers. Anrep and Starling still wrote in 1925 "a mechanical rise in blood pressure in the brain inhibits the vasomotor centre and stimulates the cardioinhibitory centre" (15).

Nitrous Oxide, Ether, Local Anesthesia

Laughing gas parties were common in the early 1800s and Humphry Davy, who repeatedly inhaled nitrous oxide in his experiments of 1799, even suggested its use as a general anesthetic agent. But no one deliberately gave it to prevent pain until 1844—42 years of missed opportunities for how many physicians (and benefits to their patients).

Michael Faraday suggested in 1818 that ether be used to prevent surgical pain, and ether jabs were a common form of social entertainment in the 1820s and 1840s; but no one gave it deliberately to prevent surgical pain until 1842.

Carl Koller, a young intern with his heart set on being an ophthalmologist, discovered in 1884 that cocaine was a superb local anesthetic for the eye. Yet since 1860, two years after the chemist Niemann had isolated cocaine from Peruvian coca leaves, it was well known that cocaine taken by mouth numbed the tongue. Koller's biographer, his daughter Hortense Koller Becker, wrote (16):

> In 1862 Professor Schroff, in a paper read before the Viennese Medical Society, pointed out that cocaine numbed the tongue, narrowed the peripheral arteries, and widened the pupils by its action via the bloodstream or when applied locally. Nor was he the only one to have experimented upon the eye. These facts were commented upon by Mantegazza in 1859, De Marles in 1862, the Spaniard Moréno y Maiź in 1868, and by many others. In 1879 von Anrep, at the Pharmacological Institute at Würzburg, wrote a comprehensive experimental paper in which he also described the locally numbing effects of cocaine and even the dilation of the pupil upon local application, and he suggested that this drug might some day become of medical importance. "Strange enough," commented G. F. Schrady in an editorial in the Medical Record of November 8, 1884, "Anrep did not note that the conjunctiva was insensible, or if so did not appreciate the significance of this fact."
>
> In the textbook on pharmacology which my father studied at the University, he had underlined the following passage which appears in the article dealing with the coca plant:
>
> "Local effects: Injection under the skin as well as painting the mucous membrane, for example, the tongue—brings about the loss of feeling and pain. 15 minutes after painting it Anrep was incapable of distinguishing sugar, salt and sour at the treated spot. Even the needle pricks could no longer be felt there, whereas the other unpainted side reacted normally. The loss of sensibility lasted between 25 and 100 minutes.
>
> "[The article concludes with] Therapeutic Uses: Up to now cocaine has not found any medical use. But on account of its powerfully stimulating effects on the psyche, respiration, and the heart, and also on account of its anesthetizing effect upon the mucous membrane, it might deserve experimental trial in quite a number of diseases. [Relative to the therapeutic use of the coca

> leaves:] There have been some experiments but no trustworthy ones over an extended period. They are, however, sold commercially and highly recommended for all possible needs."

Sigmund Freud had carried out what is still regarded as the classic pharmacological study of cocaine and the young Koller was closely associated with Freud in some of his work. Koller's daughter gives this account of why it was Koller, and not previous workers, who discovered that cocaine had great potential as a local anesthetic:

> This is the chain of events which actually placed cocaine in my father's hand and focused his attention on it: Freud's interest in the drug, awakened primarily by the American literature on substituting it for morphine, by which method he hoped to help his suffering friend, Fleischl; the actual purchase of the scarce, expensive product [by Freud] and the request he made of my father to engage in experiments during the course of which my father was required to take it by mouth. These were the circumstances that prepared the way for his particular discovery, yet cocaine had been handled, taken by mouth, and its effect even upon the eye, observed for twenty-five years without its usefulness in surgery occurring to anyone. "Upon the one occasion," my father said, "another colleague of mine, Dr. Engel, partook of some [cocaine] with me from the point of his penknife and remarked, 'How that numbs the tongue.' I said, 'Yes, that has been noticed by everyone that has eaten it.' And in the moment it flashed upon me that I was carrying in my pocket the local anesthetic for which I had searched some years earlier. I went straight to the laboratory, asked the assistant for a guinea pig for the experiment, made a solution of cocaine from the powder which I carried in my pocketbook, and instilled this into the eye of the animal." The young assistant in Stricker's laboratory, Dr. Gaertner, was the sole witness to my father's discovery ... he retold it in a 1919 newspaper of which he was medical editor:
>
> "Now it was necessary to go one step further and to repeat the experiment upon a human being. We trickled the solution under the upraised lids of each other's eyes. Then we put a mirror before us, took a pin in hand, and tried to touch the cornea with its head. Almost simultaneously we could joyously assure ourselves, "I can't feel a thing." We could make a dent in the cornea without the slightest awareness of the touch, let alone any unpleasant sensation or reaction. With that the discovery of local anesthesia was completed. I rejoice that I was the first to congratulate Dr. Koller as a benefactor of mankind."

Koller's daughter continued:

> My father was, of course, aware that local anesthesia had more general implications and was not by any means limited to operations on the eye. "I had started from the fact that the drug made the lips and tongue numb, but I limited myself to the eye, wishing to make a contribution to ophthalmology and also wishing to establish a claim to the much-coveted position an assistant at one of the large eye clinics. I did, however, directly suggest to my friend, Jellinek [assistant to Schrötter in the laryngological clinic], that he make experiments on the nose, pharynx, and larynx. He reported the results at the same meeting of the *Gesellschaft der Ärzte* (October 17) at which I read my [second] paper.

It was Koller's intense desire to obtain this position in Vienna and his conviction that an important contribution to ophthalmology would almost guarantee an offer that led him to solve the most serious problem in that specialty—how to operate painlessly and safely on the eye. General anesthesia was unsatisfactory because the patient often retched and vomited in the recovery period and because his conscious cooperation was frequently necessary during the operation. Ironically, because of strong anti-Semitism in Vienna, he never received the assistantship and, in 1888, he emigrated to America where he became one of New York's most eminent ophthalmologists, showered with many honors.

Oral Diuretics

Oral diuretics as a class are one of the two most important new groups of drugs in the last 35 years—the other being chemotherapeutic and antibiotic drugs. Not many remember that sulfanilamide led to the discovery of chlorothiazide, the first of the oral diuretics.

In 1937, Hamilton Southworth at the Johns Hopkins Hospital noted that two of 50 patients treated with sulfanilamide began to breathe deeply. As a result, he studied 15 consecutive patients treated with sulfanilamide; the blood of each showed evidence of acidosis though none had clinical symptoms of acidosis (17). Pharmacologists at Hopkins then showed that a large single dose of sulfanilamide administered to dogs produced acidosis, and Strauss and Southworth demonstrated that in three normal human subjects, sulfanilamide led to acidosis, diuresis and increase in the renal excretion of sodium and potassium (18) (see figure 2).

Diuretics had long been an important part of treatment of congestive heart failure and edema but the only really effective diuretics in 1937 were mercurial diuretics and these required intravenous injection. Was sulfanilamide an effective oral diuretic that could at last replace intravenous injections of the organic mercurial compounds? And how did it produce diuresis?

In 1933, Roughton had isolated a new enzyme, carbonic anhydrase, from red blood cells and showed its property of greatly accelerating the reaction:

$$H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow H^+ + H\cdot CO_3.$$

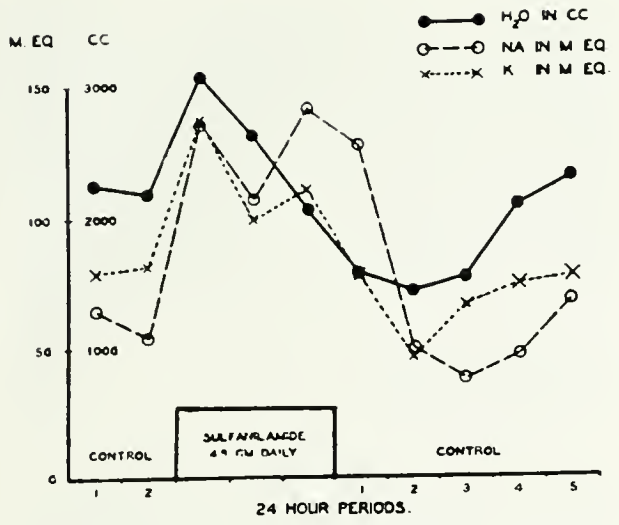

Fig. 2. After a control period of 2 days, Strauss and Southworth gave a subject 4.5 g sulfanilamide daily. Note diuresis and increased excretion of sodium and potassium.

In 1940, Mann and Keilin found that some sulfonamides inhibit carbonic anhydrase; in 1941, Davenport and Wilhelmi found that carbonic anhydrase was present in the kidney, and, in 1945, Pitts and Alexander demonstrated the role of carbonic anhydrase in the exchange of hydrogen and sodium ions in the renal tubules and in the acidification of urine. Circumstantial evidence (though later shown not to be trusted) pointed to inhibition of carbonic anhydrase as the key to diuretic agents Novello, Sprague, Beyer, and associates in the Renal Program at Merck, Sharp and Dohme synthesized a number of compounds in the sulfanilamide-carbonic anhydrase series and tested them for diuretic activity. They hit the jackpot with chlorothiazide (a compound resulting from ring closure of chlorodisulfamylaniline); it *is* a sulfonamide derivative, it *is* a carbonic anhydrase inhibitor, but its powerful diuretic action is *not* due to its ability to inhibit carbonic anhydrase (19).

Did the Hopkins group in 1937-1938 anticipate the train of events set into motion by Southworth's careful clinical observations? Dr. Robert Dripps asked this question of Southworth in 1972 and Southworth replied in part (20):

> I have been back over the original papers and have tried to think back over those days. Working with Perrin Long and Johns Hopkins, I was in on the first excitement of sulfa drugs and was lucky enough one day to pick up the first case of acidosis associated with the use of sulfanilamide. [see (17)]. The subsequent work done with Margaret Strauss of the Biochemical Department was an attempt to work out why the acidosis developed and incidentally showed that large doses did increase the urine volume and the renal excretion of sodium and potassium [see (18)]. It also showed, interestingly enough, that some hemoconcentration was produced but Bob Loeb, who went over the paper with me at Presbyterian where I had moved at the end of '37, was suspicious of the figures on blood sodiums and would not let me publish them. No one knew then about carbonic acid anhydrase and we therefore couldn't explain the acidosis. By the time this paper came out, I was in the practice of medicine and was just winding up what I had done as a resident.
>
> It is hard to remember just how much we did think of the diuretic possibilities of what we had found. I don't think it was very much and yet the paper went through the hands of Warfield Longcope and Robert F. Loeb so I was not the only one who failed to see the potential future significance. It is my vague recollection that we didn't take it too seriously because we were giving bid doses (up to 6 to 8 gms) of a drug the toxic and allergic effects of which were just being described all around us, and which we only considered using in people who had quite serious infections. I don't, however, remember any of us considering that an analogue of the drug might be developed which would have this effect in smaller doses and with less toxicity. Also those were the days when diuresis was not considered as important as it was subsequently. We had Salyrgan and we thought it was quite good for the waterlogged cardiac.

Southworth missed the opportunity to develop or participate directly in the development of oral diuretics; "It was hard," he said in retrospect, "to realize that I was on the brink of something important [in 1937-38] and didn't really appreciate it." However, his astute clinical observations showed Beyer and his associates where to look. And that must be satisfying in itself.

Other Missed Opportunities

Karl Landsteiner won the Nobel Prize for discovering human blood groups and could have won it twice over for discovering the viral cause of poliomyelitis and his work on the Rh factor. But he never connected his finding of blood groups in man with the cause of human transfusion reactions.

Werner Forssmann catheterized his own heart but missed the opportunity to follow up and use his new tool for diagnostic purposes. He tried (21) but got crushed by the German geheimrat system.

Marie Krogh devised a single breath test of diffusing capacity of the lungs but never used it for clinical diagnosis of disorders of the lungs or pulmonary capillary bed (22). However, she designed her test only to decide whether oxygen crossed the alveolar-to-capillary barrier by diffusion alone or in part by secretion. She had no need for it thereafter and in any case simple and rapid methods of gas analysis had to be invented before the test lent itself to use on large numbers of subjects and patients.

Waksman had a number of opportunities to discover strep-

tomycin before 1944. The first was early in his scientific career; his main interest was then in soil (how it decomposed organic material added to it) and not in microbes, even though he did do studies on *Streptomyces griseus* between 1916 and 1919. Again, in 1932 he missed an opportunity when his pathologist friend, Beaudette, brought him a culture of tubercle bacilli apparently killed by a fungus growing on it: Waksman failed to study it. Then in the early 1940s his son Byron, now a prominent scientist in his own right but then a medical student, wrote to his father (23):

> In reading the reprints you sent me, I was struck again with the urge to do some work in the direction of finding an effective in vivo antagonist to the tubercle bacillus. I was particularly impressed with the relative simplicity of the method you have used in isolating fungi-producing antibiotic substances, and I wondered if exactly the same method could not be used with equal ease to isolate a number of strains of fungi or actinomycetes which would act against *M. tuberculosis*. They could be tested after isolation against some more rapidly growing organism such as *M. phlei* in vitro, and finally against the tubercle bacillus itself in vivo. ...From the little reading I have done, it is my impression that no one as yet has published any work of this nature. There is no question that it has a great deal of practical value or would have if successfully concluded.

His father answered: "The time has not come yet. We are not quite prepared to undertake the problem." I wonder if this was a misunderstanding: the *son* was prepared to tackle the research while still a medical student; the *father* was not. In 1944, the father was prepared and isolated streptomycin (24).

Gross was the first to successfully ligate a patent ductus arteriosus in a child but when Taussig offered him the first crack at the now famous "blue-baby operation" he had no interest in it, and turned her down. Learning that she was thinking of moving to Boston, he advised her not to come where she would not be tolerated. "Stay where you are wanted," he said (25). She did, and so Gross left the field wide open for Alfred Blalock at Hopkins who eagerly collaborated with Taussig.

Hunt and Taveau in 1906 published a classic paper on the extraordinary physiologic activity of acetylcholine (26). In it they studied a number of other choline derivatives including succinylcholine. They completely missed its neuromuscular blocking effect because they worked on animals *curarized* at the onset of the experiment. As a result, succinylcholine never entered medical practice until 1949. Was this a missed opportunity for use of muscle relaxants in the practice of medicine and anesthesiology? Maybe not, for these reasons: (1) few surgeons or anesthetists in the early 1900s knew how to maintain artificial ventilation in man, (2) the deliberate paralysis of skeletal muscles, including muscles of respiration, probably had to await the development of a new specialty of professional, medically trained anesthetists (in the 1940s), and (3) Meltzer and Auer in their experimental studies of insufflation respiration in dogs (27) routinely gave their animals an intravenous injection of curare sufficient to abolish any spontaneous or reflex movements. Though they stated that the dog's "life is as safe [with curare] as under regular artificial respiration," they never proposed its clinical use, nor did anyone else until 1942. I doubt that availability of a pure agent, such as succinylcholine, would have made surgeons any more comfortable about operating on a paralyzed patient.

Diphenylhydantoin (dilantin) was used for 20 years to decrease the excitability of cells in the motor cortex of epileptic patients before Leonard first used it to control ventricular tachycardia in man (28).

Local anesthetics were used experimentally for 48 years to reduce cardiac excitability in animals before James Southworth and associates finally used lidocaine to reverse ventricular fibrillation in man (29). Plenty of time for missed opportunities over these two decades, especially since it encompassed the important decades in which cardiac surgery began and matured.

Ganglionic blocking agents were known as early as 1915, when Burn and Dale clearly recognized that tetraethylammonium was a ganglionic "paralyzing" agent capable of blocking sympathetic nerve impulses to the heart and arterioles. But it was not until 1946 that Lyons and associates (30) used it to lower blood pressure in hypertensive patients by performing a "chemical sympathectomy." Were there missed opportunities for its use during this 31-year period? Probably "yes" in the 1930s and thereafter, but probably "no" before, because most physicians believed that patients with hypertension had initial renal disease and reduced renal blood flow. They believed that their resulting hypertension was compensatory or essential to maintain renal circulation and prolong the patient's life, even though it eventually caused cardiac hypertrophy and heart failure.

Concluding Remarks

Much has been said and written about lags between initial scientific discovery and final clinical application. In many instances, the lag was unavoidable because whole new branches of science had to come into being before the initial discovery could move forward (e.g., the invention of the microscope could not lead to antibiotics until it first led to the discovery of microbes, the germ theory of disease and the sciences of microbiology and pharmacology). Sometimes, however, an individual made observations crucial to an important next or even final step but didn't realize their significance. These "missed opportunities" cannot be labeled scientific discoveries "locked up" in a laboratory by an unconcerned scientist. They are an almost inevitable result of the background, training and environment of that scientist and his immediate goals and preoccupations that blind him to a new concept. It has been said that the eye often sees only that behind it and

not what is in front of it. If it is any consolation to those who missed magnificent opportunities, remember that the world's greatest inventor, Thomas Edison, failed to see any useful application at all for his greatest scientific discovery, the "Edison effect," which later was the basis for the audion or vacuum tube that permitted the development of radio. And missed opportunities are not limited to science: Winston Churchill said, "Men occasionally stumble over the truth, but most of them pick themselves up and hurry off as if nothing had happened."

Julius H. Comroe, Jr.

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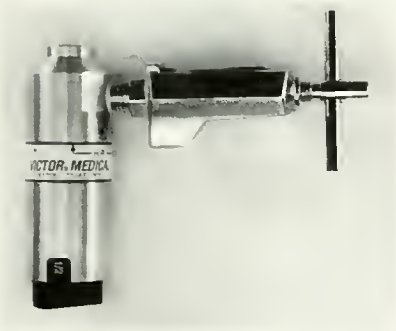

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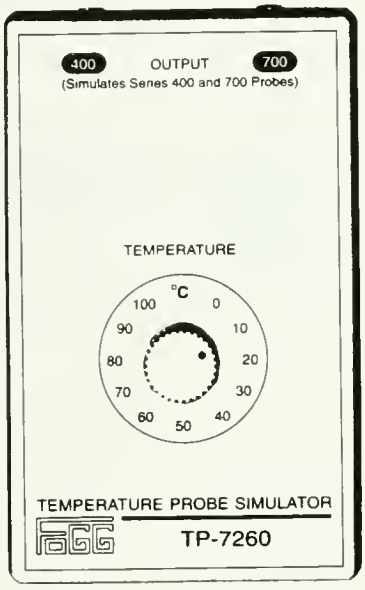

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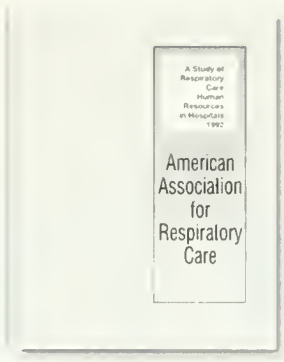

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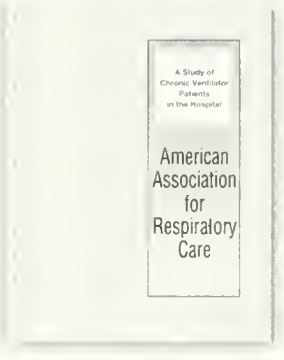

Chronic ventilator-dependent patients are costing American hospitals more than $9 million per day according to this Gallup study conducted for the AARC. This important study provides information on patients who depend on life-support systems; why, how, and where they are being treated; and the cost of treatment. 47 pages, 9 tables, 12 figures.

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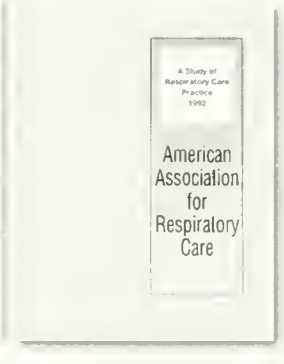

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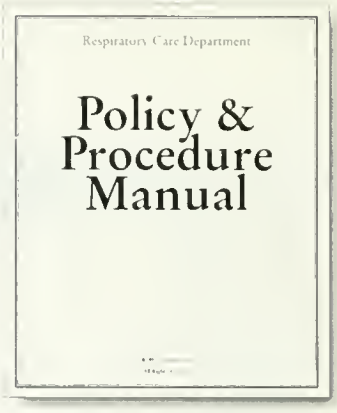

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Calendar of Events

AARC & AFFILIATES

June 13–15—California Society
28th Annual Meeting, "Through the Looking Glass: A View into the Future," at the Monterey Marriott Hotel and the Monterey Conference Center, Monterey, California.
Contact: John C Bigler CSRC Executive Director, 2763 West Ave L, Suite 164, Lancaster CA 93536, (805) 723-1180, fax (805) 723-0031.

June 19–21—Oklahoma Society
31st Annual State Seminar and Exhibition, "RCP: The Next Generation," at the Radisson Inn, Oklahoma City, Oklahoma. Golf Tournament scheduled for Wednesday, June 19.
Contact: Mike Taylor at (405) 366-3705.

July 9—AARC Video Conference
"Capturing Respiratory Services Outside the Hospital Part I: Home Care," Part 4 of the 1996, 7-part video conference, "Professor's Rounds in Respiratory Care," from 12:30 to 2 PM Eastern Time. Live telephone question-and-answer session airs July 29.
CRCE: 1 credit hour.
Contact: AARC at (214) 243-2272.

July 17–19—Texas Society
Annual Convention, "Upgrade Your Memory," at the Wyndham Anatole Hotel, Dallas, Texas.
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Contact: TSRC at (214) 680-2454.

OTHER MEETINGS

June 26–29—ECMO Programs
5th European Extracorporeal Support Organization Conference, Stockholm, Sweden.
Contact: the Congress Secretariat at +46 8 612 6900
or fax +46 8 612 6292.

September 18–21—Costa Rica Society for Respiratory Therapy
2nd Latin America Congress and the 4th Central America and Caribbean Congress for Respiratory Therapy, "Advances in Respiratory Therapy" in San José, Costa Rica.
Contact: Isabel Mesen-Sequeira, Administrative Assistant, PROJECTHOPE, SJO 078, PO Box 025369, Miami FL 33102, 011-506-290-4242, fax 011-506-296-3531.

September 19–21—ECMO Programs
8th Extracorporeal Life Support Organization Conference at the Ritz Carlton, Dearborn, Michigan.
Contact: Phoebe Hankins at (313) 998-6600.

October 23–25—ECMO Programs
10th Western ECMO Conference, Rancho Mirage, California.
Contact: Janie Waggoner of the Loma Linda ECMO Program at (909) 478-8781.

November 10–14—Asia Pacific Association for Respiratory Care
5th Congress of APARC at the Bangkok Convention Centre and Central Plaza Hotel, Bangkok, Thailand.
Abstracts must be submitted by July 31.
Contact: Congress Secretariat, 5th Congress of APARC, Congress and Convention Experts (Thailand) Co Ltd, PO Box 10-163, Ladphrao, Bangkapi, Bangkok 10310, THAILAND.

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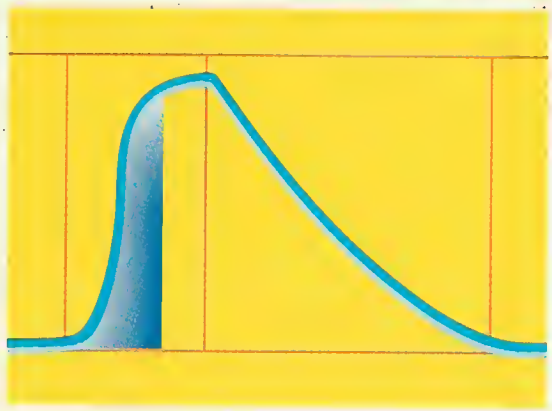

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